“CICATRICIAL ALOPECIA - CLINICAL, DERMASCOPIC AND HISTOPATHOLOGICAL STUDY”

Dissertation submitted in partial fulfilment of the Requirements for the degree of

M.D.(DERMATOLOGY, VENEREOLOGY & LEPROSY)

BRANCH XX

DEPARTMENT OF DERMATOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI-600 003

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

MAY 2018

CERTIFICATE

This is to certify that the dissertation titled **"CICATRICIAL ALOPECIA - CLINICAL, DERMASCOPIC AND HISTOPATHOLOGICAL STUDY"** is a bonafide work done by **Dr.Priyadharsini J,** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2015– 2018. This work has not previously formed the basis for the award of any degree.

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DECLARATION

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DECLARATION

I, **Dr. Priyadharsini J ,** solemnly declare that this dissertation titled **"CICATRICIAL ALOPECIA - CLINICAL, DERMASCOPIC AND HISTOPATHOLOGICAL STUDY"** is a bonafide work done by me at Madras Medical College during 2015-2018 under the guidance and supervision of **Prof. U. R. DHANALAKSHMI, M.D., D.D., D.N.B.,** Professor and Head of Department, Department of Dermatology, Madras Medical College, Chennai-600003.

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CONTENTS

Introduction

INTRODUCTION

Cicatricial Alopecia is a scarring form of Alopecia which occurs due to permanent destruction of hair follicles leading to patchy hair loss. It was first described by Brocq in 1885.There is damage to the stem cell niche in the bulge region of hair follicle which destroys any potential for hair regrowth.

It can be either due to Primary cause affecting the follicles or by an external process (Secondary Alopecia).

The most common causes of primary cicatricial alopecia includes Lichen plano pilaris, Chronic cutaneous lupus erythematosus, Pseudopelade of Brocq in the descending order of frequency. Other rare causes include Central centrifugal cicatricial alopecia, Alopecia mucinosa, Keratosis pilaris , spinulosa declavans, Folliculitis declavans, Dissecting cellulitis, Acne keloidalis , Erosive pustular dermatosis, Frontal fibrosing alopecia and non specific end stage cicatricial alopecia.

Secondary causes of cicatricial alopecia may be following trauma, secondary to sclerosing disorders, granulomatous condition, neoplasm, developmental defects, hereditary disorders and infections like Cutaneous Tuberculosis etc.

Patients with cicatricial alopecia usually have complaints like itching, burning, scaling, discharge and scarring leading to considerable disfigurement of their scalp which can be a major psychosocial burden for them. Hence early diagnosis, identification of the causative factors and prompt treatment is crucial .

Dermascopy is a noninvasive mode of diagnosis to identify scalp disorder. Dermascopy reveals loss of follicular ostia. Other features seen are follicular pustules and scattered single hair follicle. Characteristic findings may be seen in some conditions like DLE which shows Patchy brownish discoloration with thick arborizing blood vessels and Lichen plano pilaris showing predominant feature of peripilar cast.

Histopathological study serves as a confirmatory diagnostic tool. It helps in differentiating cicatricial and non cicatricial alopecia.Also amongst cicatricial alopecia based on the predominant infiltrate they may be further classified as lymphocytic and neutrophilic.

Though the etiological and pathogenesis of cicatricial alopecia have been evaluated very few data are available on published literature. Dermascopy being a non invasive tool may aid in early diagnosis and in certain conditions can limit the need for biopsy. The concordance between the findings of dermascopy and histopathology can also be appreciated in this study.

Scalp hair is a significant cosmetic element and prompt diagnosis and aggressive management is crucial in preventing permanent disfigurement. Hence this study is conducted to emphasize on the early diagnostic features based on clinical, dermascopic and histopathological findings and identifying the underlying cause of cicatricial alopecia

Review of Literature

REVIEW OF LITERATURE

Cicatricial alopecia consists of group of hair disorders in which hair folicle is irreversibly destroyed leading to permanent alopecia.[1]

Anatomy of hair follicle

Scalp hairs are mainly terminal hairs. They are long , coarse, pigmented with larger diameters . The hair follicles are typically arranged in the follicular unit composed of one to four terminal hairs and one to two vellus hairs, sebaceous gland, and encircled by the same arrector pili muscle[2]. Each hair grows steadily, approximately 1 cm per month and continuously for 3–5 years (anagen phase). Growth then stops and is followed by a brief catagen phase and a 2–4-month telogen phase, during which old hair is shed. With the onset of the anagen phase, new hair starts to grow from the same follicle[1]. Scalp hair has a diameter of 0.017 to 0.18 mm and its exterior consists of a layer of flat, imbricated scales pointing outward from root to tip.

A mature hair follicle may be divided histologically into Upper and lower segments[2].

Upper segment consists of infundibulum extending from the follicular ostium to sebaceous duct entrance. Isthmus extends from sebaceous duct above to the attachment of arrector pili muscle below.

Lower segment consists of the stem and the bulb[2]. It extends from the arrector pili muscle attachement to the base of hair follicle below. (figure 1)

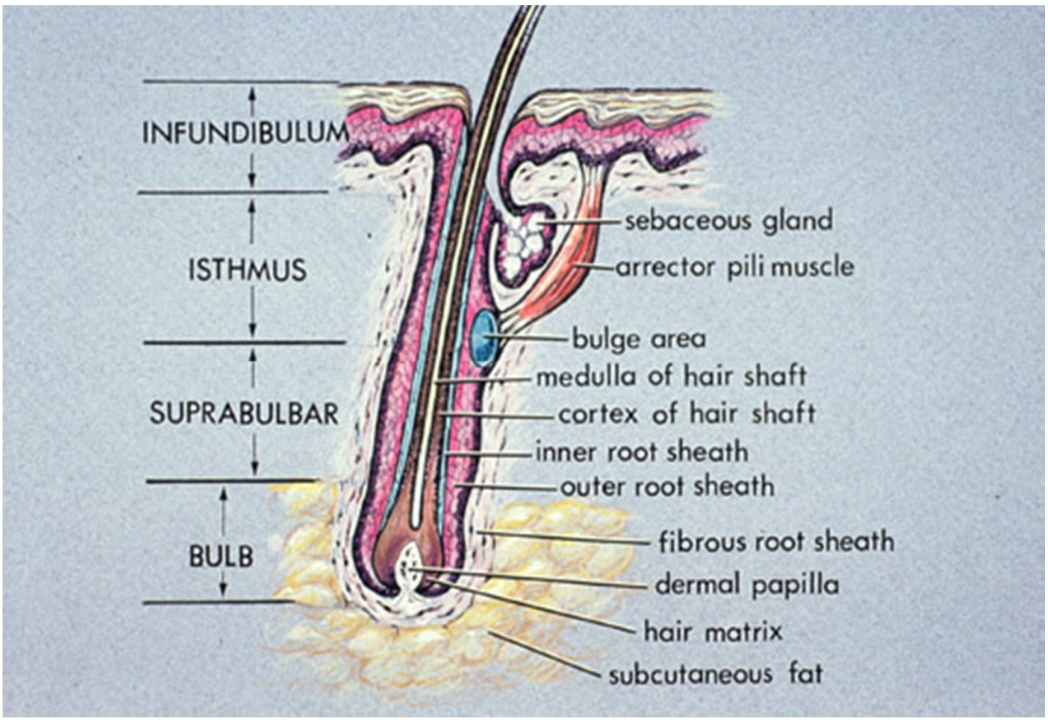


Figure 1: Structure of hair follicle

Infundibulum:

It is funnel shaped. Acrotrichium is the portion at which it passes through the epidermis[2]. Very little glycogen is present in infundibular epithelium. Infundibular epithelium is identical to epidermis with which it is continuous and cornifies in same manner. A granular zone with numerous kerato hyaline granules are present.[3] Basal layer lacks rete ridges.

Isthumus:

The inner root sheet desquamates and is completely lost at this portion. The outer root sheet begins to cornify[4,5].

The stem or suprabulbar region:

This is the longest section of hair follicle in anagen growth phase. Extends from base of the isthumus to the summit of the bulb. From outside inward it is composed of an outer root sheath ,an inner root sheath , cuticle of hair, cortex and medulla[2]. Adamsons fringe is where the inner root sheet begins to cornify. (figure 2)

Bulb:

This is the lowest part of the hair follicle. It resembles bulb of an onion. It constitutes epithelial matricial cells amongst which melanocytes are interspersed. The expanded base encloses a follicular papilla which is continuous with peri follicular connective tissue[2]. The peri follicular sheath has an outer longitudinally arranged bundle of collagen and inner bundle of collagen that encircles the follicle. (figure 2)

The bulb consists of the outer root sheath, Henle and Huxle layer of inner root sheath followed by the cuticle of inner sheath, cuticle of the hair, cortex , medulla[6] from outside to inside.

Perifollicular connective sheath:

It surrounds the entire follicle[2].it is different from the connective tissue sheath that surrounds the infundibulum and isthmus

The hair:

It represents complete maturation of follicular metrical cells. It has a cortex and inner medulla. The medulla is often discontinuous or absent. The medulla contains melanosomes[2], empty vacuoles and citrulline rich granules. Keratin filaments of cortex have plenty of disulphide bonds which provides its tensile strength. Colour of hair depends on amount and distribution of melanosomes within it.

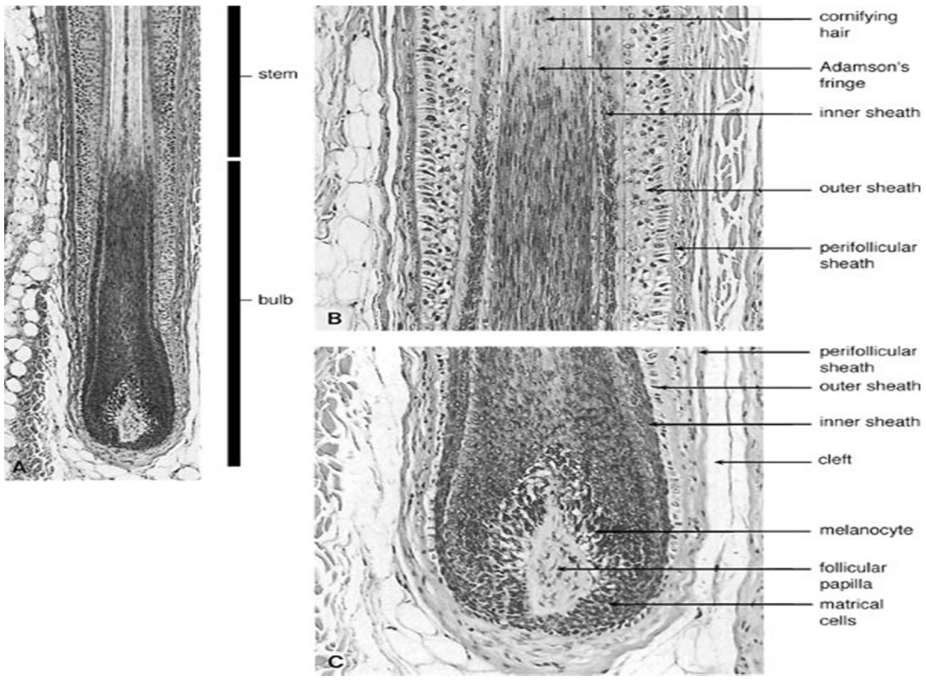


Figure 2: Bulb and part of stem of hair follicle.

Hair cycle:

A complete replication of matrix cells of follicle on human scalp is 39 hours of duration. The three phases of a follicle are (figure 3)[2]

1. Anagen
2. Catagen
3. Telogen

Anagen phase :

It is an active growth phase. It can be divided into six stages (I–VI) and anagen I–V (proanagen) anagen VI (metanagen)[7]

In proanagen phase hair progenitor cells proliferate, envelope the growing dermal papilla, grow downwards into the skin, and begin to differentiate into the hair shaft and inner root sheet; then, the newly formed hair shaft begins to develop. In anagen VI (metanagen) the melanocytes located in the hair matrix show pigment producing activity.There is full restoration of the hair fiber-producing unit, which is characterized by formation of the epithelial hair bulb surrounding the dermal papilla, located deep in the subcutaneous tissue, and the new hair shaft appears from the skin surface. This phase can last for several years in hair follicles.[7]

The catagen phase:

It starts when the anagen growth phase comes to the end. At the beginning of the catagen phase, differentiation and proliferation of hair matrix keratinocytes

decreases significantly, the pigment-producing activity of melanocytes stops, and hair shaft production is completed.[2] The hair follicle undergoes apoptosis-driven regression resulting in a reduction of about one-sixth of the normal diameter. During catagen, a specialized structure, the club hair is formed. The epithelial lining is identical to that of isthumus of outer root sheath. The dermal papilla is transformed into a cluster of quiescent cells closely adjacent to the regressing hair follicle epithelium and travels from the subcutis to the dermis/subcutis border to maintain contact with the distal portion of the hair follicle epithelium, including the secondary hair germ and the bulge. This phase lasts for a few weeks.[8]

The telogen phase:

It begins after the catagen phase; the hair goes into a resting phase, and this period can last few weeks (eyelashes) to eight months (scalp hair). Although the hair does not grow during this stage, the dermal papilla stays in the resting phase. Telogen hair follicles are characterized by a lack of pigment-producing melanocytes and the IRS. Their dermal papilla is closely attached to a small cap of secondary hair germ keratinocytes containing hair follicle stem cells. Approximately 10–15% of all hairs are in resting phase at any given moment[3]. At the end of this stage, the hair falls (exogen phase); a few weeks later, the hair follicle re-enters the growth phase by stimulating stem cells from the bulge area.[5]

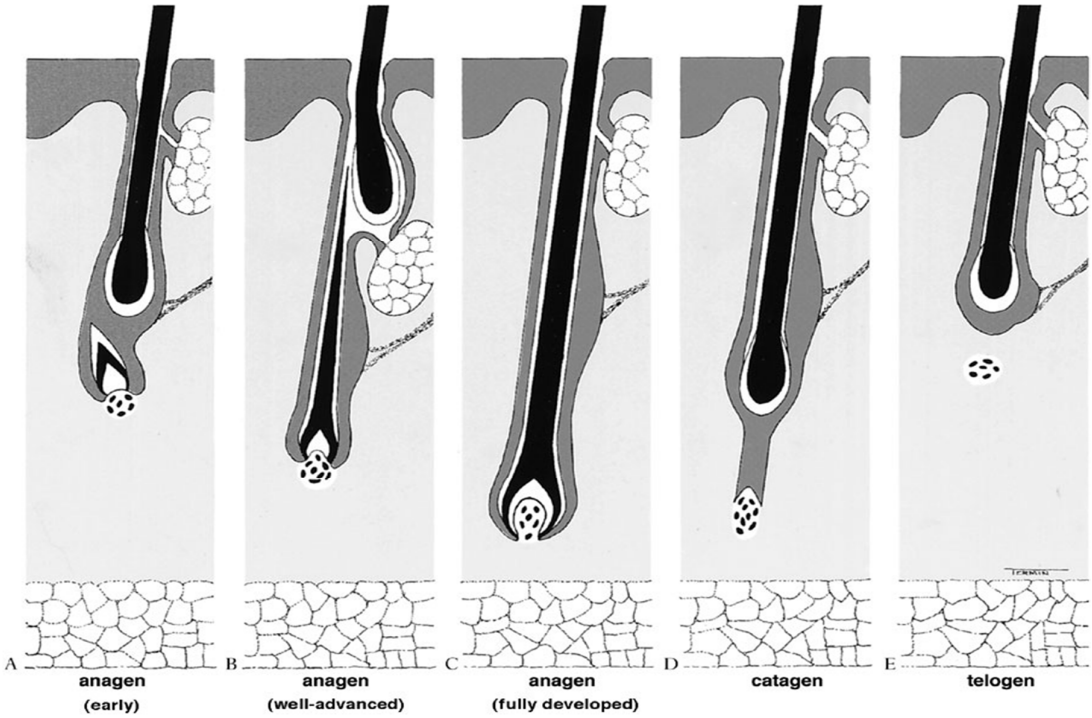


Figure 3: Hair cycle

Vascular supply:

Cutaneous vascularization is provided by arterioles that enter the subcutaneous fat and ascend into the dermis forming a plexus that supplies cutaneous structures such as the hair follicle. At the lower portion of the hair follicle they form a rich vascular network [2,19]. In anagen there is an increase of perifollicular vascularization correlatedwith the upregulation of vascular endothelial growth factor expression by keratinocytes of ORS.

Nerves include sensory afferents and autonomic sympathetic nerves. They rise from the dermis or subcutaneous tissue and ascend from the dermal network to innervate the hair follicle from the bulb to the epidermis.[10] At the level of the sebaceous gland, hair follicles are surrounded by a collar of nerves, frequently

called the hair end organ, which is arranged in an outer circular layer and an inner longitudinal layer. There is a second primarily horizontal dermal plexus located at the junction of the papillary and reticular dermis. Nerve branches extend from this network, primarily in association with vessels, to innervate the papillary dermis and epidermis.

Cicatricial alopecia

Cicatricial alopecia is an enigmatic group of hair disorder with destruction of hair follicles leading to permanent hair loss[1]. Histologically, the follicles are replaced by fibrous stellae.[4]

Epidemiology

Brocq in 1885 described cicatricial alopecia[11] . It later became known eponymously as pseudopelade of Brocq , which is now regarded as a separate entity. Quainquad[12] described folliculitis decalvans, a form of scarring alopecia in which pustular folliculitis of the advancing margin was a conspicuous feature.

Primary cicatricial alopecias are usually seen around third to fourth decade of life. The racial and sexual variations depend upon the type of cicatricial alopecias.

Pathophysiology[13]: (fig.4)

- Genetic factors
- Environmental factors
- Hair follicle immunology

- Loss of CD 200 expression
- Collapse of immune privilege
- Cytotoxic T cell mediated damage
- Pro inflammatory response
- Increased Apoptosis in Primary Cicatricial Alopecia
- Sebaceous Gland Dysfunction
- Peroxiosome Proliferation Activated Receptor gamma Deficiency
- Programmed cell death
- Neurogenic Skin Inflammation

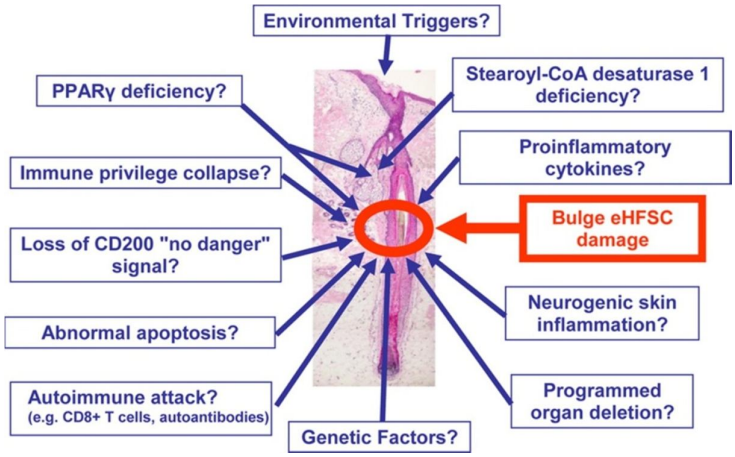


Figure 4. Pathogenesis of primary cicatricial alopecia[13]

Genetic factors

X -linked and autosomal-dominant modes of inheritance have been demonstrated in keratosis follicularis spinulosa decalvans[14]. Graham Little syndrome was found to have human leukocyte antigen DR1[15] on human leukocyte antigen typing. These may show some relation between genetic factors and PCA.

Environmental Triggers

- Infection[16,17]
- Trauma[18]
- medication [19]

Hair follicle immunology:

The distal hair follicle is rich in langerhan cells and T cells. Hair follicle immune system controls and suppresses hair follicle inflammation.. The anagen hair bulb has an established area of Immune privilege. This area lacks MHC class molecules and also possess intra follicular cortisol thus providing immune suppression activity. In primary cicatricial alopecia expression of bulb immune privilege is collapsed. The hair follicle bulge immunostains with CD8/144B antibody which recognizes cytokeratin 15 in keratinocytes[13]. The bulge cells have high proliferative capacity and express b1 integrin In cicatricial alopecia there is insult to this follicular bulge hence leading to permanent alopecia.

Collapse of immune privilege:

The follicle bulge is an important niche for keratinocyte stem cell [13]. Collapse of immune privilege exposes the bulge stem cells(fig 6) to various pro inflammatory cytokines(fig.5)

Loss of CD 200 expression:

Cd200 plays a role in anti inflammatory action an absence of which leads to inflammatory response and destruction of hair follicle.[13,20]

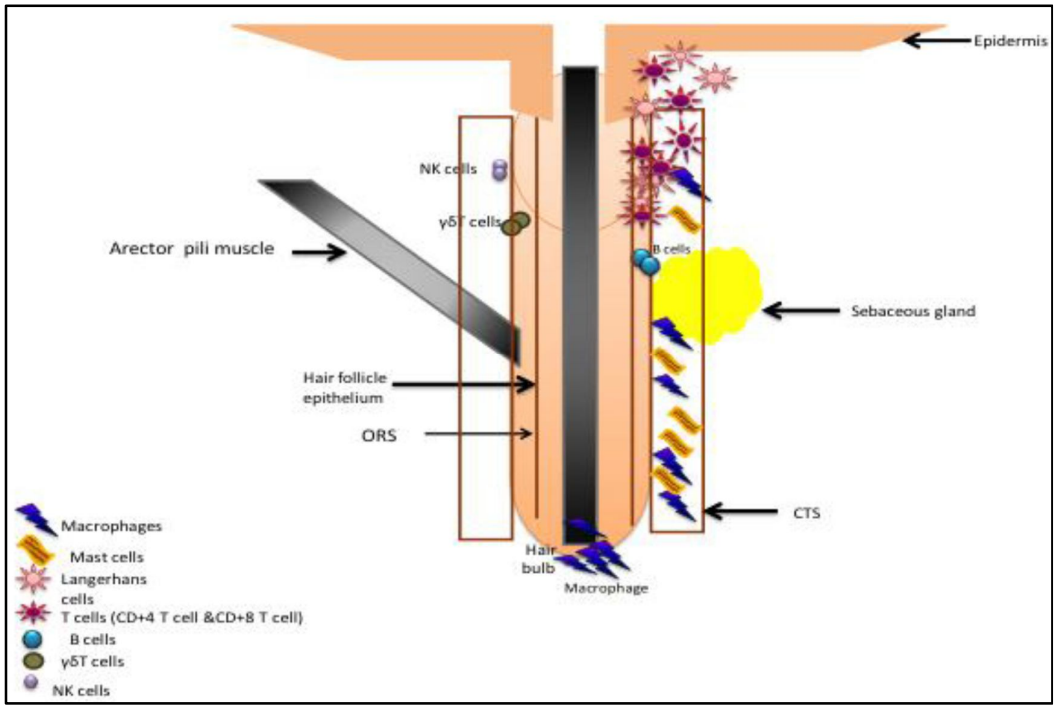


Figure : 5

Cytotoxic T cell mediated damage[13]:

There is increased expression of cytotoxic T cells[21] in peri follicular area which may also explain its role in cicatricial alopecia Cell surface and cell to cell adhesion molecules are increased resulting in trafficking of immune cells

Pro inflammatory response:

There is Th1 pattern of inflammation with production of INF gamma, INL2 and TNF a[22]. In older lesions fibroblast growth factor and TGFb[23] gets upregulated resulting in scarred tissue.

Increased Apoptosis in PCA:

P53 gene is upregulated[13] in chronic cutaneous lupus erythematoses and lichen plano pilaris. Epidermal apoptosis is increased in cicatricial alopecias which may be mediated through Fas-Fas ligands[24,25]. Apoptosis inhibitor bcl 2 is decreased in cicatricial alopecia.

Sebaceous Gland

A defect in stearoyl-CoA desaturase 1, important in sebaceous gland fatty acid composition, results in sebaceous glands atrophy and abnormal gland secretions, in turn leading to delayed inner root sheath disintegration, retrograde hair shaft growth, and penetration of the bulb, with resulting foreign body reaction and eventual destruction of the hair follicle[26].

PPAR Deficiency

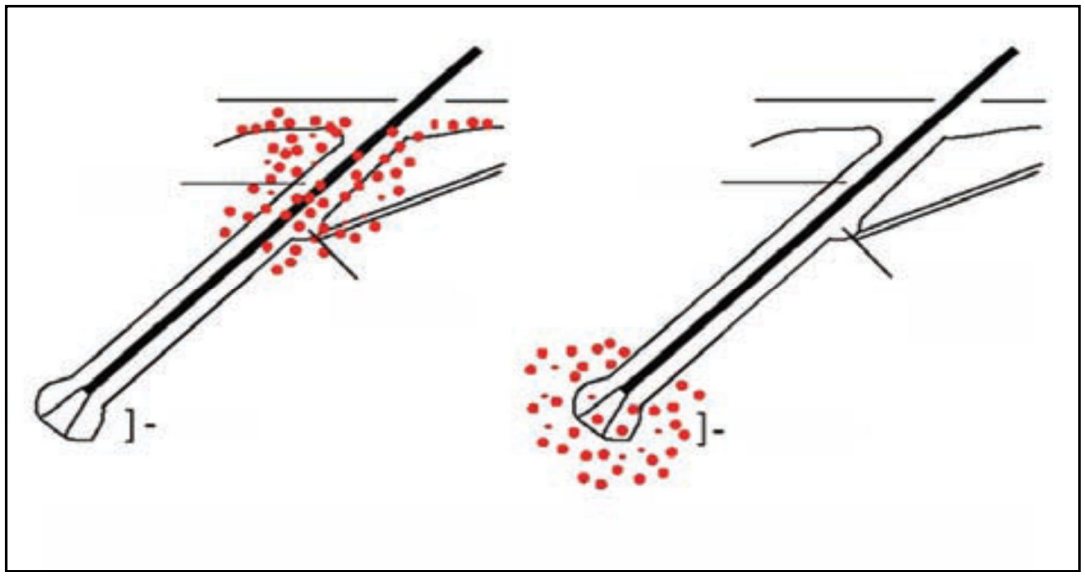
Decreased expression of peroxiosome proliferator activated receptor gamma [13]was found in the hair follicles affected by lichen plano pilaris. This is required for regulating lipid metabolism and peroxisome biogenesis[27]. Hence there is progressive loss of peroxisomes, proinflammatory lipid accumulation and infiltration of inflammatory cells followed by destruction of the pilosebaceous unit.

Programmed cell death

There is increase in FAS ligand[13] mediated apoptosis of hair follicle cells.

Neurogenic Skin Inflammation

Psycho-emotional stress can induce nerve growth factor (NGF) which increase the release of substance P (SP), which induces mast cell leading to peri-follicular inflammatory cell infiltrates[28] .

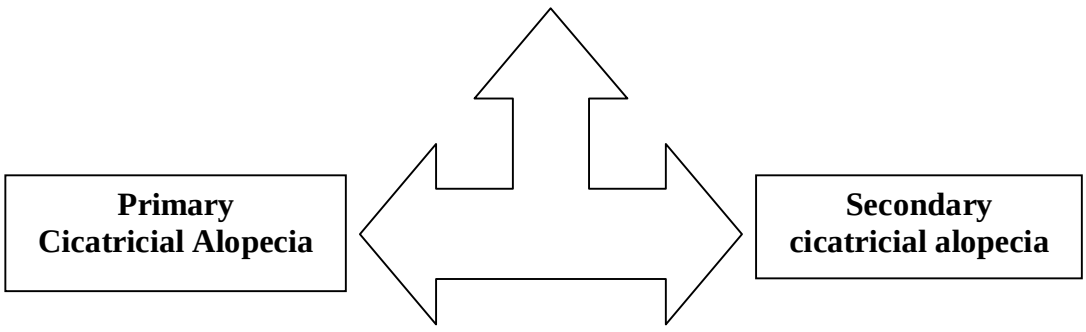


Cicatricial alopecia Non cicatricial alopecia

Figure 6[29]: difference between cicatricial and non cicatricial alopecia

Classification of cicatricial alopecia[1]

CICATRICIAL ALOPECIA



PRIMARY CICATRICIAL ALOPECIA CLASSIFICATION

Lymphocytic[1]	**Neutrophilic**[1]	**Mixed**[1]	**Non specific or end stage cicatricial alopecia**[1]
Chronic cutaneous lupus erythematosus	Folliculitis decalvans (including tufted folliculitis)	Acne keloidalis	
Lichen planopilaris (LPP) Classic LPP Graham-Little syndrome Frontal fi brosing alopecia	Dissecting cellulitis / folliculitis	Acne necrotica	
Pseudopelade of Brocq		Erosive pustular dermatosis	
Central centrifugal cicatricial alopecia			
Alopecia mucinosa			
Keratosis pilaris spinulosa decalvans[30]			

CLASSIFICATION OF SECONDARY CICATRICIAL ALOPECIA[30]

- Traumatic
- Sclerosing disorders
- Granulomatous
- Infectious
- Neoplastic
- Developmental defects and hereditary disorder

TRAUMATIC CICATRICIAL ALOPECIA

- Radiodermatitis
- Mechanical trauma
- Postoperative (e.g. fl ap necrosis)
- Burns
- Accidental alopecia
- Dermatitis artefacta
- Traction alopecia
- Hot comb alopecia

Sclerosing disorders

- Morphoea
- Scleroderma
- Lichen sclerosus
- Sclerodermoid porphyria cutanea tarda
- Chronic graft-versus-host disease

Granulomatous Sarcoidosis

- Necrobiosis lipoidica
- Infectious granulomas

Developmental defects and hereditary disorders

- Aplasia cutis
- Facial hemiatrophy (Romberg's syndrome)
- Epidermal naevi
- Hair follicle hamartomas
- Incontinentia pigmenti
- Focal dermal hypoplasia of Goltz
- Porokeratosis of Mibelli
- Ichthyosis
- Epidermolysis bullosa
- Polyostotic fi brous dysplasia
- Conradi–Hünermann syndrome (chondrodysplasia punctata)

Infections [32]

Bacterial	**Fungal**	**Viral**	**Protozoal**
Folliculitis	Kerion	Shingles	Leishmaniasis
Carbuncle/furuncle	Favus	Varicella	
Mycobacterial Tuberculosis	Tinea capitis (rarely scarring)	HIV	

Neoplastic

Benign	Malignant
Cylindroma	Primary Basal cell carcinoma
Other adnexal tumours	Squamous cell carcinoma Cutaneous T-cell lymphoma
	Secondary Renal, breast, lung, gastrointestinal Carcinoma.
	Lymphoma, leukaemia

Stable and unstable cicatricial alopecia[33]

Stable	Unstable
Burns	Cutaneous discoid lupus erythematosus
Radiation-induced	Classic lichen planopilaris
Prior hair transplantation	Frontal fi brosing alopecia
Prior rhytidectomies and brow lifts	Graham-Little syndrome
Traction alopecia	Classic pseudopelade of Brocq
Trichotillomania	Alopecia mucinosa
Congenital aplasia cutis	Neutrophilic alopecia
Central centrifugal cicatricial alopecia	Keratosis follicularis spinulosa decalvans

CLINICAL FEATURES OF CICATRICIAL ALOPECIA

CHRONIC CUTANEOUS LUPUS ERYTHEMATOSES:

Women to men ratio is 2 : 1. More commonin African Americans. The incidence is approximately 1 in 2000. The peak age of onset is around 40 years. Characterised by plaques of erythema [34]and scaling with follicular plugging and scarring. Itching may be present

Pseudopelade of Brocq[35]

Occurs in both sexes . Common in middle age. It always remains confined to the scalp. The affected patches are smooth, soft and slightly depressed. At an early stage in the development of any individual patch there may be some erythema. The patches tend to be small and round or oval, but irregular bald patches may be formed by the confluence of many lesions. The course is extremely variable. Most often there is slow development over many years of small round patches of alopecia that ultimately converge to produce larger irregular areas of hair loss.

Diagnostic criteria for pseudopelade of Brocq. (Braun-Falco et al)[35]

Clinical criteria

- Irregularly defined and confluent patches of alopecia
- Moderate atrophy (late stage)
- Mild perifollicular erythema (early stage)
- Female : male ratio 3 : 1

- Long course (more than 2 years)
- Slow progression with spontaneous termination possible

Direct immunofl uorescence

- Negative (or only weak IgM on sun-exposed skin)

Histological criteria

- Absence of marked inflammation
- Absence of widespread scarring (best seen with elastin stain)
- Absence of significant follicular plugging
- Absence, or at least decrease of sebaceous glands
- Presence of normal epidermis (only occasional atrophy)
- Fibrotic streams into the dermis

CLASSICAL LICHEN PLANUS[36]

The onset is between 30 and 70 years Characterised by violaceous papules, erythema and scaling. These papules are replaced quickly by follicular plugs and scarring. Eventually, the plugs are shed from the scarred area, which remains white[36], smooth and atrophic. Follicular orifices are absent within the area of alopecia.

FRONTAL FIBROSING ALOPECIA

It typically occurs in post menopausal women . Its a clinical variant of LPP. There is loss of follicular orifices, and perifollicular erythema and hyperkeratosis at the marginal hairline. The frontal hairline recedes in a straight line rather than bitemporally.

Graham little syndrome

Reported by Graham-Little in 1915**.** It is known eponymously and variously as the Graham-Little, Lassueur–Graham-Little or Piccardi–Lassueur–Little syndrome [37]Most patients are women between the ages of 30 and 70 years. Characterized by progressive cicatricial alopecia of the scalp, loss of pubic and axillary hair without clinically evident scarring, and the rapid development of keratosis pilaris .

Central centrifugal cicatricial alopecia:

Characterized by slowly progressive scarring of vertex that spreads symmetrically and centrifugally, alopecia is incomplete with islands of unaffected hairs within scar area[33]. Peri-follicular hyperpigmentation and polytrichia can be observed. Usually asymptomatic but unusual sensations, such as pins and needles, itch or tenderness may occur.[38]

Alopecia mucinosa:

Characterized by follicular papules coalescing to form erythematous well-defi ned, indurated [38]plaques with prominent follicular opening with loss of hair[33] .

Keratosis follicularis spinulosa decalvans[33]:

Characterized by widespread erythematous follicular hyperkeratosis followed by punctate atrophy and cicatricial alopecia [38]

NEUTROPHILIC CICATRICIAL ALOPECIA

Folliculitis decalvans/Tufted folliculitis

Caused by Staphylococcus aureus. Presents as suppurative and destructive folliculitis that are painful and pruritic[33] Clinically characterized by erythematous pin-point follicular pustules, papules with boggy swelling with dolls hair appearance[39]. It extends peripherally. Ultimately leads to crusting and scarring in the centre .

Dissecting cellulitis of scalp:

They present as suppurative painful fluctuant nodules and abscesses. There are interconnecting sinuses with multifocal lesions[33] . These lesions eventually coalesce to give cerebriform appearance to scalp. Along with acne conglobata, hidradenitis suppurativa and pilonidal sinus, it forms the follicular occlusion triad/tetrad

Mixed group

Acne keloidalis nuchae:

Commonly seen on occipital scalp and nape of neck . They are Pruritic , painful firm follicular papules and pustules[33]. They coalesce to form large nodules . They heal with hypertrophic scars or keloidal plaques[40]

Acne necrotica varioliformis:

Characterized by crops of pruritic, tender, erythematous, umblicated papulo-pustules that undergo central necrosis with resultant scar formation the sites involved are anterior scalp, eyebrows, nose, neck and chest[33].

Erosive pustular dermatosis:

Characterized by chronic relapsing amicrobial pustular dermatoses, progresses to form well-demarcated boggy swelling, which is easily deroofed to reveal beefy red, exudative Erosions[33].

Investigations

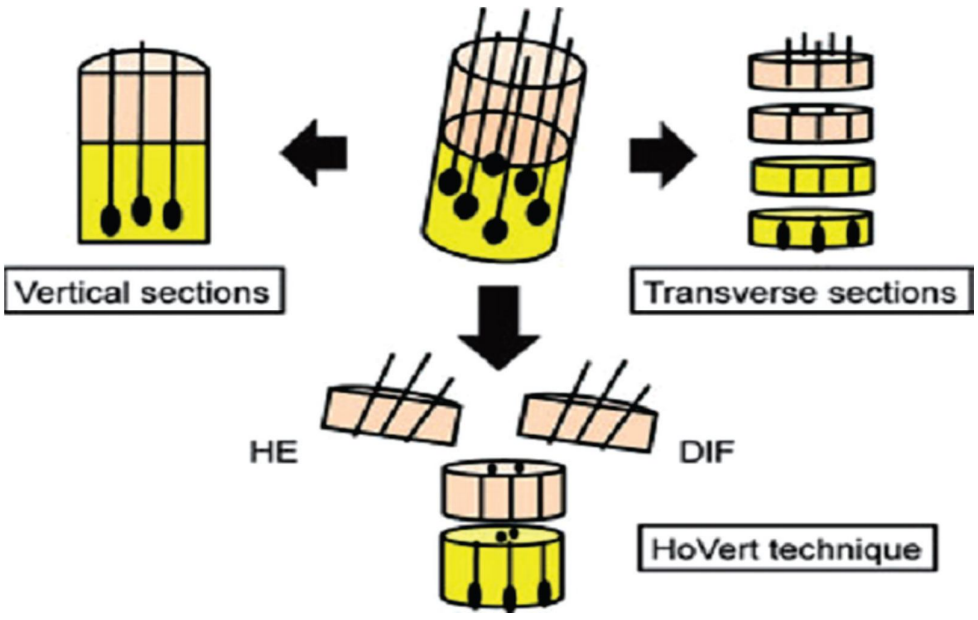
- **Hair pull test**
- **Dermascopy**
- **Scalp biopsy and Histopathology study**
- **Immunofluorescense**
- **Confocal microscope**
- **Microarray analysis**

Hair pull test

It is a crude test. Approximately 60 hairs are gathered between thumb and forefinger of non dominant hand . With the dominant hand thumb and forefinger grasp the hair near the scalp and gently pull it. The number and type of hair extracted give clue to the diagnosis. In cicatricial alopecias of primary type there is increase in extraction of anagen hair.

Scalp biopsy:

4mm punch biopsies are taken from the scalp and sent for vertical and horizontal section. It is stained with eosin and hematoxin stains . Special stains may be employed if necessary.



Histopathological features of various types of cicatricial alopecias[41]

CCLE[33]

Epidermis may be atrophic. There is interface dermatitis with periadnexal, perifollicular ,perivascular and interstitial lymphocytic infiltrate .Dermis shows deposition of mucin. There is atrophy of sebaceous glands and follicular plugging. Scattering of dyskeratotic keratinocytes are noted[38].

Lichen planopilaris

'Active' stage shows follicular plugging,interface dermatitis with dense band like lymphocytes obscuring DEJ. Cytoid bodies seen in basement membrane[43].

Absent sebaceous glands with or without pigmentary incontinence noted. Saw toothed rete ridges are seen. 'longitudinal tracts' of fibrosis at the site of former follicle with peri-follicular lamellar fibrosis and adjacent epidermal atrophy is seen in end-stage

Pseudopelade of Brocq:

Early stage shows variable amount of perifollicular lymphocytic infiltrate , followed by eccentric atrophy of follicle infundibular epithelium[38]. Later stages there is concentric lamellar fibrosis around hair follicle and fibrosing follicular tract.

Elastin stains reveal dense elastic tissue cuffing[38]

Central centrifugal cicatricial alopecia[33]

Earliest feature is premature desquamation of inner root sheath. In later stages lamellar fibrosis and lymphocytic inflammation surround the hair follicle leading to follicular destruction and fibrous tract formation[38]

Alopecia mucinosa

Mucinous degeneration of the Outer root sheeth and sebaceous glands seen A perifollicular lymphocytic infiltrate with eosinophils and histocytes is found

Keratosis follicularis spinulosa decalvans

Compact hyperkeratosis, hypergranulosis of the upper follicular layer with superficial intrafollicular and peri-follicular edema seen in the early stage. In advanced stage there is concentric perifollicular, horizontal adventitial lamellar Fibrosis with scarred follicular tracts

Folliculitis decalvans/tufted folliculitis:

Acneiform dilatation initially with perifollicular neutrophilic infl ammation around the upper follicle, which later develops into a more mixed infl ammatory infiltrate of neutrophils, lymphocytes, and plasma cells[40]. Follicular rupture ensues, resulting in foreign-body giant cell granuloma formation seen around exposed hair shaft fragments. In later stage, follicular and adventitial fi brosis is seen.

Dissecting cellulitis of scalp:

Infundibular acneiform distention with prominent intrafollicular and perifollicular neutrophilic infi ltration, with abscess formation on follicular rupture composed of neutrophils,lymphocytes and plasma cells[43] seen. In later stages abscess become partially lined by squamous epithelium forming sinus tracts.

Acne keloidalis nuchae:

Early disease there is perifollicular and intrafollicular lymphoplasmacytic infiltrate more pronounced in sebaceous glands, in advanced stage there is complete follicular destruction occurs with prominent loss of sebaceous glands and dermal fi brosis[44].

Acne necrotica varioliformis:

In early disease there is well defined dense perivascular and perifollicular lymphocytic infiltrate with prominent sub-epidermal edema[45]. Necrosis of the individual keratinocyte is seen initially and is followed by the confluent necrosis of the central follicle and interfollicular epidermis.

Erosive pustular dermatosis:

Nonspecifi c, variable degree of epidermal erosions, atrophy, acanthosis, parakeratosis, and sub-corneal pustules. The dermis shows mixed chronic infl amatory infiltrate with reduced or absent hair follicles[33].

DERMASCOPY IN CICATRICIAL ALOPECIA

DLE[46]

- Atrophy
- Loss of follicular openings
- Arborizing telangiectasia.
- Prominent hyperkeratotic follicular plugging is seen at the periphery
- Dark brown to blue-grey pigment pattern in interfollicular area[47]

LPP [48]

- Peripilar casts,
- Target “blue-grey dots[48]
- White dots in chronic stage

Pseudopelade of Brocq

- Diagnosis of exclusion[46]

Keratosis Folliculitis Spinulosa Declavans[46]

- Black dots
- Follicular keratosis
- Short cut-off hairs
- Hypotrichosis
- Perifollicular scales
- Honeycomb appearance

Central Centrifugal CA [48]

- Honeycomb pigmented network
- terminal hairs
- vellus hairs
- peripilar white halo
- pin-point white dots
- white patches
- erythema
- scales
- asterisk-like brown blotches
- broken hairs
- dark peripilar halo.

Dissecting cellulitis[47]

- Yellow dots, appearing as 3D structures imposed over dystrophic hairs.
- Advanced cases are diffi cult to differentiate from
- Other scarring alopecias

Folliculitis decalvans

- Multiple upright hairs (>5) emerging from a single ostium[47],
- Follicular scaling is seen
- Follicular pustules are seen at the active border.
- Twisted capillary loops may be seen in the interfollicular region.
- These vascular patterns may not be well-appreciated in the darker pigmented skin type

- The inactive scarred areas are seen as pinkish-white patches with absent follicular openings.

Seborrheic Keratosis

- Milia, Fat fingers, Crypts & Ridges, Blue grey lobules, Finger print

Direct immunofluorescence study

It is an important diagnostic tool differentiating between primary cicatricial alopecia due to LPP or DLE. In DLE, it shows granular deposits of immunoglobulin (IgG) and complement (C3) at the dermoepidermal junction[50], while in LPP there is shaggy deposition of fibrinogen and clumped IgM along follicular Basement membrane[33]

Confocal microscopy

Reflectance Confocal Microscopy (RCM) is an in vivo, noninvasive, real-time, en-face. Microscopic imaging of the superficial layers of the skin down to the superficial reticular dermis[33]. The resolution at cellular level is comparable to conventional low power microscopy . It reveals epidermal disarray, spongiosis, exocytosis of inflammatory cells in the epidermis, interface dermatitis, peri- and intraadnexal infiltration of inflammatory cells, dilated vessels in the dermis, dermal infiltration of inflammatory cells, melanophages, and dermal sclerosis as features of PCA.

Thus, RCM is a high-resolution imaging technique that may be helpful in the diagnosis and follow-up of scarring alopecia[33]. It may also help in choosing the most appropriate biopsy site for more informative histology.

Microarray analysis[51]

Microarray analysis represents the global gene expression profile, as a diagnostic tool for clinically or pathologically indistinguishable PCA. It is generated from total RNA isolates from active lesions of LPP and PPB, distinguished the two conditions, which were thought to be related

TREATMENT[38,33,52]

DLE

First line

- Topical corticosteroids
- I/L Triamcinolone acetonide injections

Second line

- Oral corticosteroids (dose1 mg/kg, for initial actively progressing disease, tapered over 8 weeks)
- Antimalarials (Hydroxychloroquine dose 200-400 mg/day)
- Retinoids (acitretin and Isotretinoin, dose10-40 mg/day)

Third line

- Topical immunomodulators 0.1% tacrolimus and 1% pimecrolimus,
- Topical 5-FU, topical tazarotene, imiquimod
- Mycophenolate mofetil
- Methotrexate
- Azathioprine

- Oral vitamin E[53]
- Oral gold
- Dapsone
- Clofazamine
- Intralesional INF alpha2 may be given
- Monoclonal anti-CD4 antibodies given

LPP

First line[54]

- Potent topical corticosteriods
- I/L Triamcinolone acetonide

Second line

- Oral corticosteroids
- Oral cyclosporine
- Topical cyclosporine
- Oral tetracycline

Third line

- Oral retinoids
- Antimalarials
- Mycophenolate mofetil
- Thalidomide
- Griseofulvin

- Eximer laser
- PPARγ agonist like thiozolidinediones
- Intralesional triamcinolone acetonide

Graham Little Picardi[55]

- Topical and intralesional corticosteroids
- Topical tacrolimus
- Systemic corticosteroids
- Oral ciclosporin

PoB

- Potent topical corticosteroids

Alopecia mucinosa[38,52]

First line

- Potent topical corticosteroids

Second line

- Intralesional steroid triamcinolone
- Tablet Minocycline
- Topical±Oral indomethacin
- Tablet Dapsone

Third line

- Systemic steroids
- Capsule Isotretinoin
- Antimalarials
- PUVA therapy
- interferon a-2b+Interferon gamma
- superficial X-rays

Keratosis Folliculitis Spinulosa Declavans[56]

- Potent topical corticosteroids±keratolytics
- Oral antibiotics
- Tablet Dapsone
- Oral retinoids
- Laser epilation

Folliculitis declavans [57]

First line:

- Oral ± topical antibiotics given

Second line

- Oral rifampicin (dose 300 mg BD)+oral clindamycin (dose300 mg BD

Dissecting cellulitis of scalp

First line

- Oral isotretinoin
- Oral isotretinoin+ (i/l) Triamcinolone acetonide

Second line

- Oral antibiotics + Topical antibiotics / Topical Retinoids
- Aspiration and (i/l) Triamcinolone acetonide

Third line

- Low dose corticosteroids
- Colchicine
- Tablet Dapsone
- Excision and skin grafting
- Lasers and radiotherapy

Acne keloidalis nuchae

First line

- Potent topical steroids [58]
- Oral antibiotics+topical steroids/ Intralesional Triamcinolone

Second line

- Surgical excision[58]
- CO2 laser
- Diode laser hair epilation

Third line

- Radiotherapy[58]
- Capsule Isotretinoin

Acne necrotica varioliformis[59]

- Oral antibiotics Oral isotretinoin
- I/L triamcinolone

Erosive pustular dermatosis of scalp[60]

- Topical corticosteroids
- Topical immunomodulators
- Calcipotriol cream
- Oral Isotretinoin

SURGICAL TREATMENT OF CICATRICIAL ALOPECIA[61]

The choice of treatment depends on

- the type of cicatricial alopecia
- availability of donor's hair
- scalp laxity
- patient's healing
- characteristics and vascular supply

Unstable cicatricial alopecia[61] is secondary to disorders that have a tendency to progress and recur intermittently over the course of time.

Therefore, it is essential to identify the type of alopecia and to confirm quiescence for at least 1‑year

The available surgical modalities are,[62]

- hair transplantation
- scar excision
- flap surgery
- scar reduction with tissue expansion

Scalp grafting

Scalp grafting of composite hair-bearing has proved to be an ingenious procedure for surgeon and patient alike because of its simplicity and versatility[63]. Flaps and reductions are preferred to grafts because of lesser number of operations and time required to obtain a good result. Scalping flaps are used for large defects not amenable to other forms of surgical treatment.

Disadvantages:

- long unsightly scars
- unappealing skin
- decreased sensation
- donordefect
- unaesthetic

Scalp reduction

It is indicated for complete or partial elimination of alopecia on the vertex of the scalp. The donor skin is provided by tissue expansion method[67] . The donor skin should match with the texture and hair-bearing characteristics. This can be achieved by recruiting local tissue, with primary closure at the donor site.

Disadvantages

- Cannot be done in case of lack of sufficient donor scalp, replacement with expanded flaps are done.

Tissue expansion

This was introduced for cicatricial alopecias with large defect[67]. It recruits both sensory nerve supply and hair-bearing tissue into the recipient site when needed.

Five factors to consider when planning a tissue expansion strategy:

1) The dimensions of tissue to be replaced
2) Proper expander selection
3) Incision type and placement for expander insertion[67]
4) The number of expanders to be used
5) The schedule for saline injections

Disadvantage

- two-stage procedure,
- multiple visits to the surgeon for repeated injections into the expander,
- infections,
- risk of skin necrosis
- high cost
- Exposure of tissue expander

. auto grafting can be followed by topical minoxidil 5% appears[64]

Psychological impact

Although cicatricial alopecia is not a life threatening condition, affected individuals may experience substantial psycho-social distress[65]. This clearly reflects the negative impact of cicatricial alopecia on the patient's psychosocial state[65].

Aims & Objectives

AIM OF THE STUDY

To determine in the cases of cicatricial alopecia the following:

1) To study the clinical pattern of the disease
2) To study the dermascopic features of the disease
3) To study the histopathological findings of the disease
4) To identify various etiological causes of the disease

Materials and Methods

MATERIALS AND METHODS

4.1Study Design:

Prospective cohort study.

4.2 Study approval :

Prior to commencement of this study - Thesis & Ethical Committee of Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai had approved the thesis protocol.

4.3 Place of study :

Rajiv Gandhi Government General Hospital

4.4 Period of study :

Duration starting from November 2016 to September 2017

4.5 Sample size :

50 cases

4.6 Inclusion Criteria

All new cases presenting with cicatricial alopecia attending dermatology OPD, Rajiv Gandhi Government General Hospital.

4.7 Exclusion Criteria

Patients with cicatricial alopecia who have been treated with either topical or systemic drugs

4.8 Methodology

This study was carried out as a clinical study of cicatricial alopecia conducted from November 2016 to September 2017. The sampling procedure is summarized as follows.All Patients attending dermatology outpatient department having cicatricial alopecia were randomly selected for the study. Sample size is fifty. Written informed consent of all the patients were taken for the study. The patients were interviewed in person. Detailed case history of each patient was taken. In clinical history questions were asked in reference to the duration of the disease, whether it was gradual or insidious in onset, its course, associated skin manifestations and other symptoms like itching, pain, scarring were noted. History of predisposing factors like trauma and infections were noted. Patients were also questioned of relevant personal and family history. Clinical examination was done and morphology of lesion including site, size and number of lesions were noted. Hair pull test was done to all the patients. Presence of hair loss in other sites and a complete dermatological examination was done to rule out presence of lesions in skin, mucosa and nails.

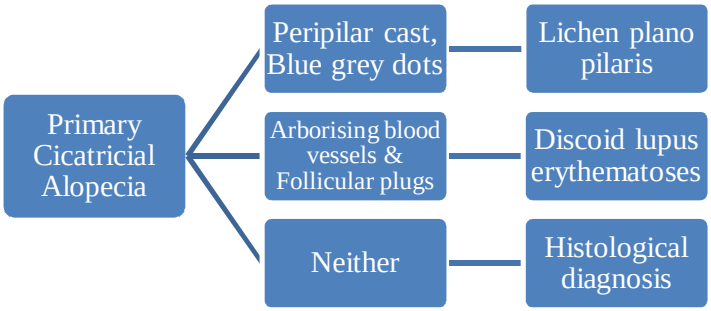
Routine investigations of all patients were performed which included a complete hemogram ,renal and liver function tests. In relevant cases,VCTC, VDRL,pus culture and sensitivity, mantoux,scraping to rule out fungal infections, Woods lamp examination and anti nuclear antibodies were done.

Dermascopic examination of the scalp was done using a non polarized trichoscope.cicatricial alopecia is characterized by absent follicular opening. The centre and peripheral margin of the alopecia patches were examined and photographs were taken. Absence of follicular ostia was considered hallmark of cicatricial alopecia. A presumptive diagnosis was made from trichoscopic features as follows

Loss of follicular ostia

⬇

Cicatricial alopecia



A scalp biopsy was done for all patients and sent for histopathological study with eosin and hematoxylin stain.

4.9 FOLLOW UP PROCEDURES:

Patients were asked to come for follow up after 1 month. The investigation reports were collected and recorded and classified. The diagnosis was confirmed on the basis of clinical, histopathological, and Dermascopy features. Patients were treated according to their relevant diagnosis.

4.10 STATISTICAL ANALYSIS:

The collected data were analysed with IBM.SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables. To find the significance in categorical data Chi-Square test was used. In the above statistical tool the probability value 0.05 is considered as significant level

4.11 ETHICAL ISSUES

Participants were made aware about the nature and purpose of the study. It was also informed to all the participants that all data provided by the patients will be kept confidential and will be used only for the study purpose. Willingness and signature of the participants were taken on a previously designed consent form. Written consents were obtained from all the subjects who participated in the study before data are collected. Detailed description of the study and the aspects of patient confidentiality are explained to the subject and voluntary participation is sought. Institutional ethics committee of Madras medical college reviewed the study proposal for ethical consideration and approval

Observations & Results

OBSERVATION AND RESULTS

TABLE 1A: AGE DISTRIBUTION

Age	Frequency	Percent
4-20 yrs	7	14
21 - 30 yrs	11	22
31 - 40 yrs	15	30
41 - 50 yrs	10	20
51 - 60 yrs	5	10
Above 60 yrs	2	4

TABLE 1B: GRAPHICAL PRESENTATION OF AGE DISTRIBUTION

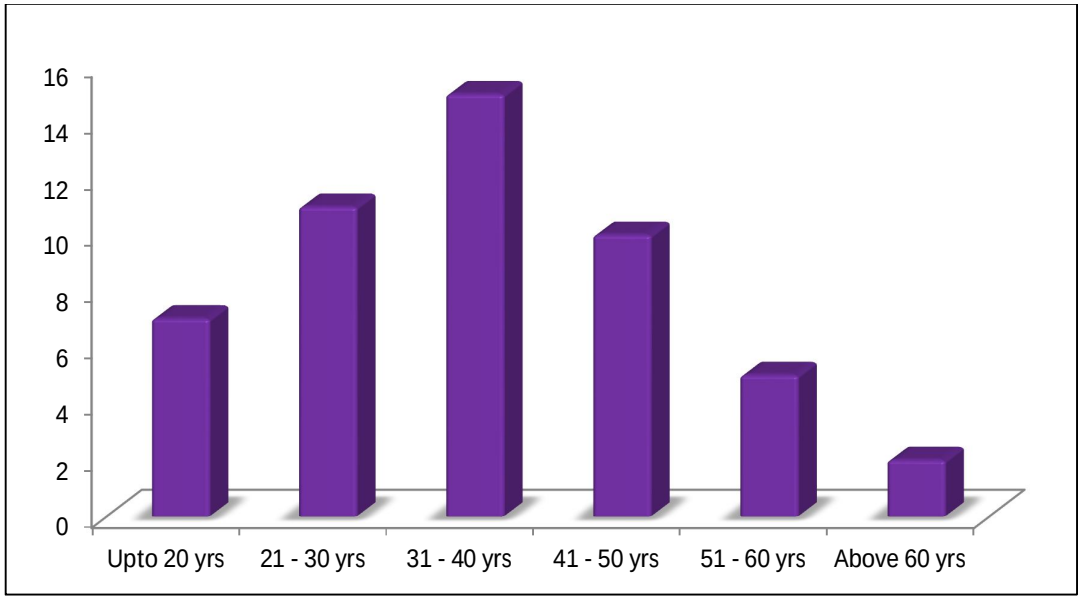


Table 1 shows that maximum number of patients with cicatricial alopecia were between 31-40 years (30%) followed by 22% patients between 21-30 years, 20% patients between 41-50 years, 14% between 4-20 years and less than 4% above 60 years.

TABLE 2.A.: SEX DISTRIBUTION OF PATIENTS

SEX	NUMBER OF PATIENTS	PERCENTAGE
MALE	22	44
FEMALE	28	56
TOTAL	50	100

TABLE 2.B. GRAPHICAL REPRESENTATION OF SEX DISTRIBUTION

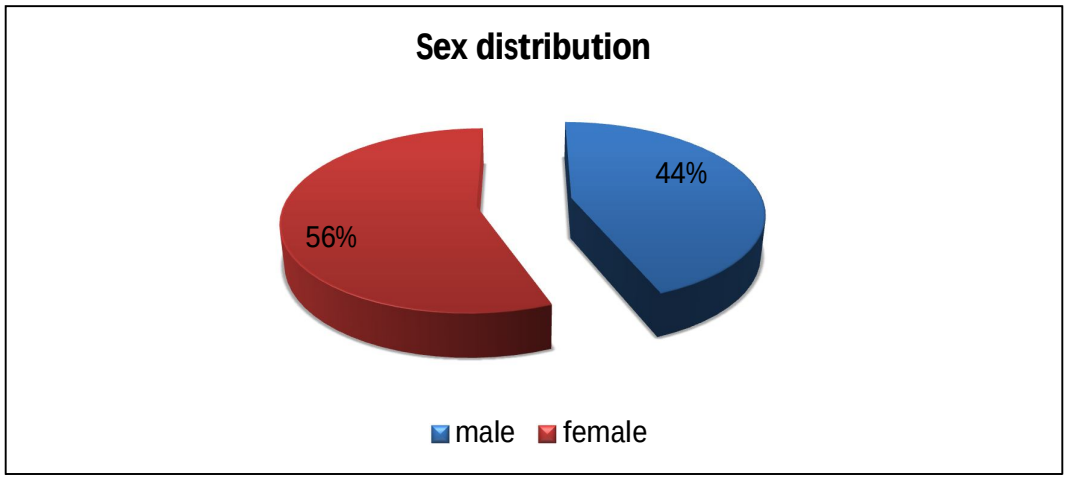


Table.2C AGE DISTRIBUTION AMONGST MALE & FEMALE PATIENTS

	Maximum Age	Minimum Age
Male	12	67
Female	4	68

Table2 shows the prevalence of the disease is common in the female population with female to male ratio1.3:1.

TABLE 3.A. DISEASE ONSET

	Number of patients	Percentage
Gradual	48	96
Acute	2	4

TABLE 3.B. GRAPHICAL REPRESENTATION OF DISEASE ONSET

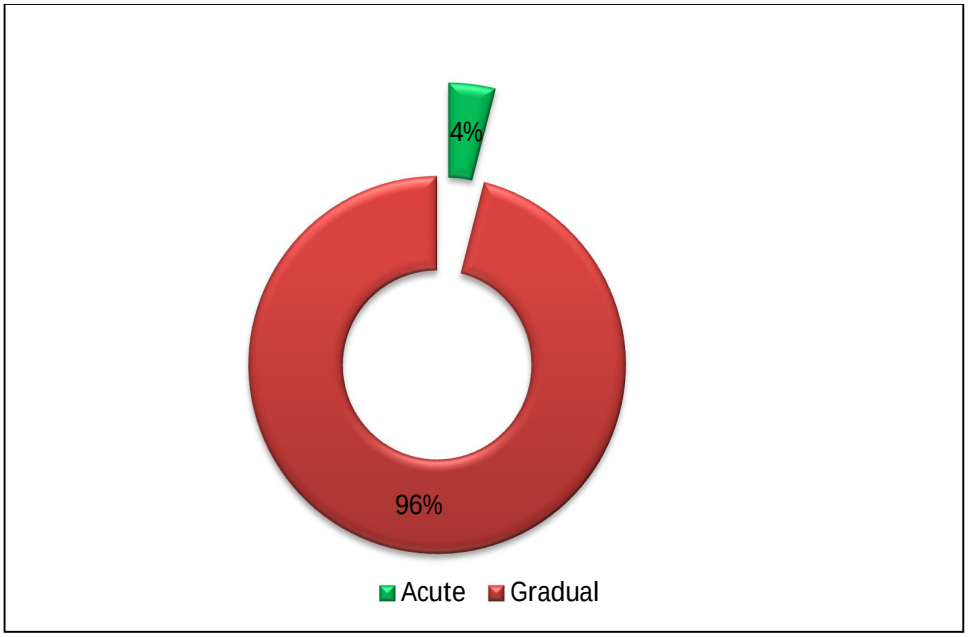


Table 3 shows that 96% percent of patients had gradual onset and 4% had acute onset of the disease. The duration of the disease ranged from 1 month to 10 years with an average of 20.1 months.

TABLE 4.A. : NUMBER OF ALOPECIA PATCHES

	Number of Patients	Percentage
Single	21	42
Multiple	29	58

TABLE 4.B. GRAPHICAL REPRESENTATION OF NUMBER OF ALOPECIA PATCHES

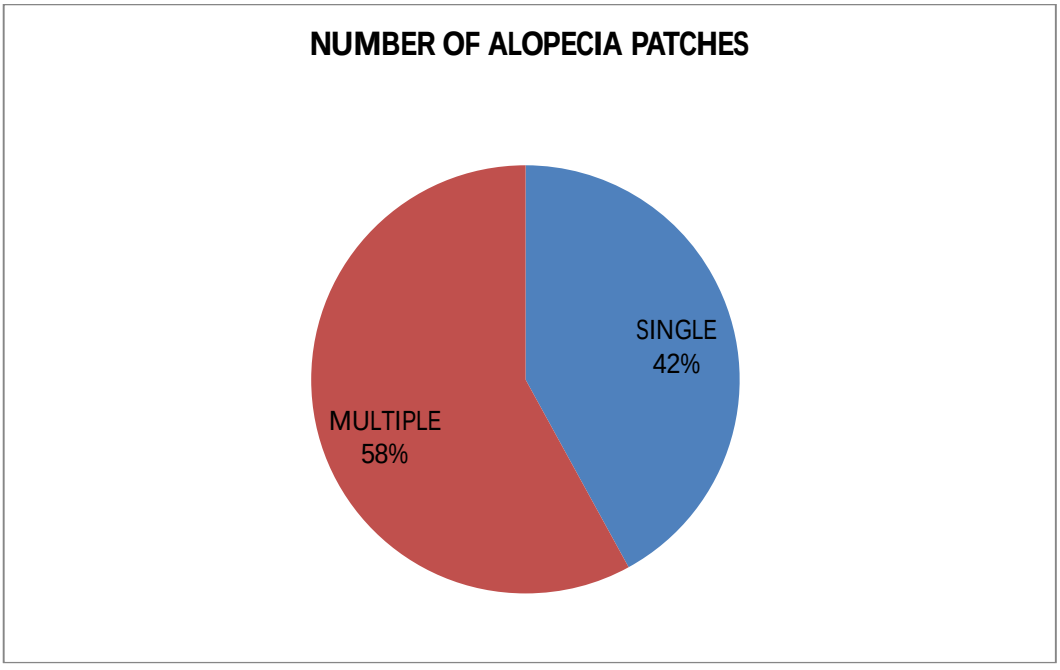


Table 4 shows that 58% patients had multiple patch and 42% patients had single patch.

TABLE 5.A MORPHOLOGY. OF ALOPECIA PATCH

	Number of patients	Percentage
Diffuse	7	14
Patchy	43	86

TABLE 5.B.GRAPHICAL REPRESENTATION OF MORPHOLOGY OF ALOPECIA PATCH

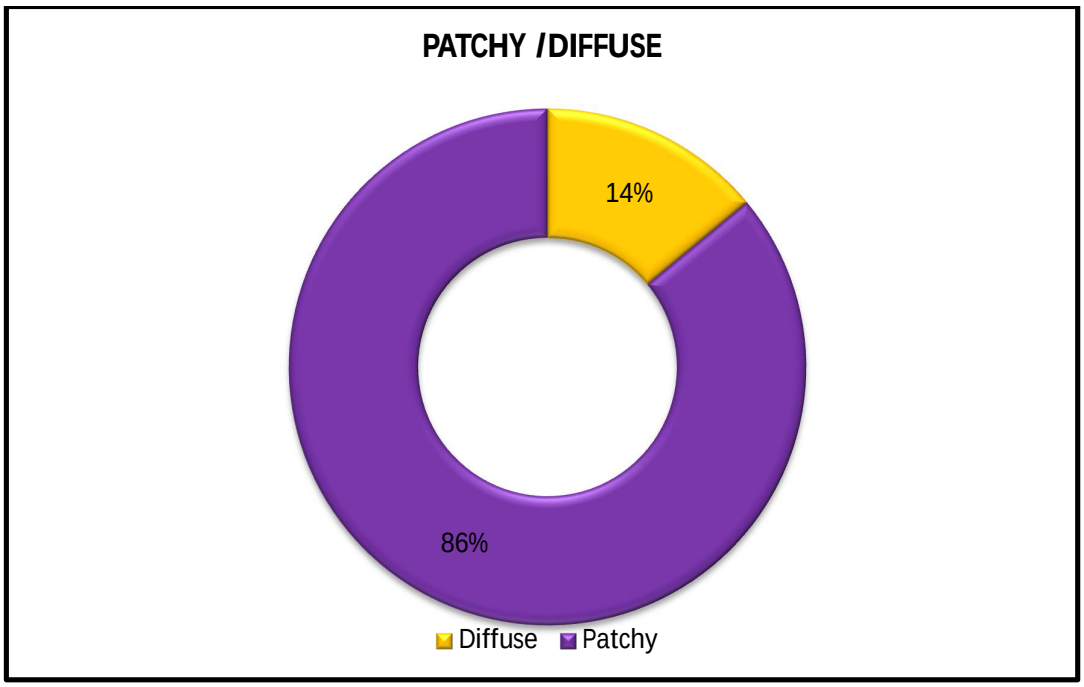


Table 5 shows 86% of patients had patchy involvement of alopecia patches in the scalp while 14% had diffuse involvement of the scalp. The most common site of involvement was found to be the vertex of the scalp with an average of 60%.

TABLE 6. GRAPHICAL REPRESENTATION OF SKIN LESIONS IN PATIENTS

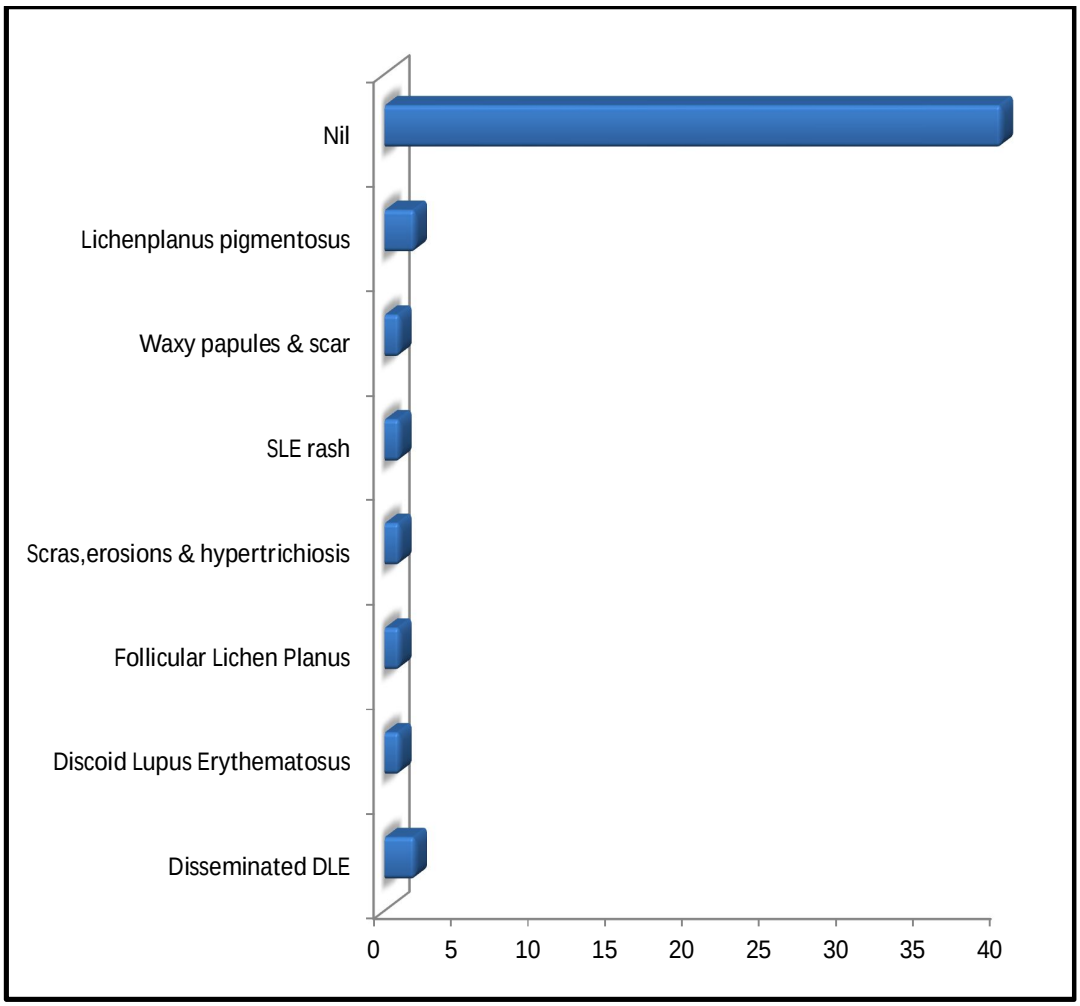


Table 6 shows the presence of skin lesions in 20% of patients with cicatricial alopecia the most common being plaques of discoid lupus erythematoses . This signifies the important of complete dermatological examination in patients with cicatricial alopecia and their association in other conditions

TABLE 7.A. ETIOLOGY OF CICATRICIAL ALOPECIA

	Number of patients	Percentage
Lichen plano pilaris	15	30.0
Post radiotherapy cicatricial alopecia	1	2.0
Post traumatic cicatricial alopecia	2	4.0
Acne keloidalis nuchae	2	4.0
En coup de sabre	1	2.0
Grahamlittle syndrome	1	2.0
Gunthers disease	1	2.0
Lipoid proteinosis	1	2.0
Naevus sebaceous	2	4.0
Seborrheic keratosis	1	2.0
Trichilemmal cyst	1	2.0
Discoid lupus erythematosus	13	26.0
Keratosis spinulosa declavans	1	2.0
Non specific Cicatricial AAlopecia	4	8.0
Pseudopelade of Brocq	3	6.0
Systemic lupus erythematoses	1	2.0
Total	50	100.0

TABLE 7.B. GRAPHICAL REPRESENTATION OF ETIOLOGY OF CICATRICIAL ALOPECIA

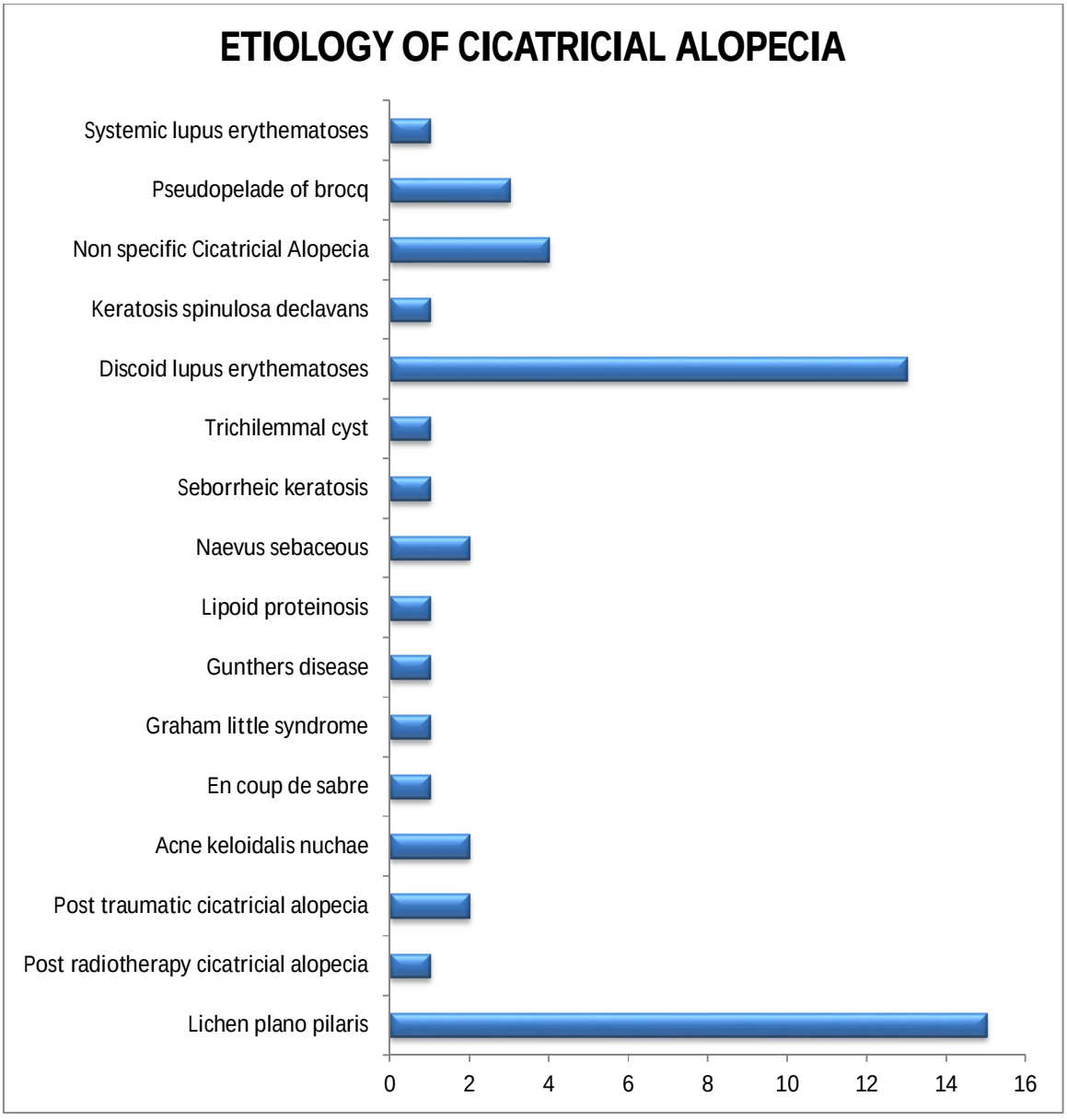


TABLE 7.C. PRIMARY AND SECONDARY CICATRICIAL ALOPECIA

PRIMARY CICATRICIAL ALOPECIA	SECONDARY CICATRICIAL ALOPECIA
Discoid lupus erythematoses = 13	Trichilemmal cyst= 1
Lichen Plano Pilaris = 15	Naevus sebaceous = 2
Graham Little syndrome = 1	Lipoid proteinosis = 1
Pseudo pelade of Brocq = 3	En coud de sabre = 1
Acne keloidalis nuchae = 2	Trauma = 2
Keratosis spinulosa declavans = 1	Radiotherapy = 1
Non specific cicatricial alopecia = 4	Gunthers disease = 1
Discoid lupus erythematosis with SLE = 1	Seborrheic keratosis = 1
Total = 40	Total = 10

N = 50.(40+10)

Table 7 shows that 80% of cicatricial alopecias are of primary causes with only 20% constituting the secondary causes. Amongst the primary cicatricial alopecia Lichen plano pilaris has the maximum number of cases (30%), followed by discoid lupus erythematoses (26%). Non specific cicatricial alopecia and Pseudo pelade of Brocq constituted 8% and 6% respectively. Amongst the less frequent causes were acne keloidalis nuchae(4%), keratosis spinulosa declavans and systemic lupus erythematoses 2% each. Amongst the secondary causes of cicatricial alopecia trauma was the most common (4%) followed by 2% each of radiation, lipoid proteinosis, Gunthers disease, and scalp tumors.

TABLE 8: CLINICAL FEATURES OF VARIOUS TYPE OF CICATRICIAL ALOPECIA

	N= 50	Age mean	Sex		Duration (yrs)	Skin lesions
			M=22	F=28		
LPP	15	39.50	11(50%)	4(14.2%)	1.04	2
DLE	13	39.38	4(18%)	9(32.2%)	4.5	3
POB	3	48.6	1(4.5%)	2(7%)	5.5	0
AKN	2	35.5	2(9%)	-	2.25	0
KSD	1	17	-	1(3.5%)	7.5	0
GLP	1	68	-	1(3.5%)	0.5	1
Sle with DLE	1	24	-	1(3.5%)	0.16	1
Non specific CA	4	30.75	2(9%)	2(7%)	3.25	0
Lipoid proteinosis	1	15	1(4.5%)	-	14	1
Naevus sebaceous	2	12.5	1(4.5%)	1(3.5%)	12.5	0
Trichilemmal cyst	1	4	-	1(3.5%)	4	0
En coup de sabre	1	18	1(4.5%)	-	4	0
Trauma / RT	3	32.3	2(9%)	1(3.5%)	11.6	0
Gunthers	1	21	1(4.5%)	-	.08	0
SK	1	67	1(4.5%)	-	10	0

Discoid lupus erythematosus

The patients include four males and nine females. The most common site was vertex in both males and females .There were multiple, erythematous, scaly, atrophic, depigmented plaque with peripheral hyperpigmentation. In 3 (23%) patients, the lesions were also present on other body areas. In one of the cases , scalp DLE was associated with systemic lupus erythematosus.

Lichen planopilaris

The patients include 4 males and 9 females. The involvement in most of the patients was on the vertex followed by parietal area. Violaceous patches of scarring alopecia with perifollicular scaling were seen. Itching was an important symptom in most of the patients(46%). One patient had oral(6%) Lichen Planus and 2 (13%) had skin lesions of Lichen planus

Graham Little Picardi

One patient presented with Violaceous Patches of scarring alopecia of the scalp involving the right parietal and vertex area. There was non scarring alopecia of the axilla and follicular lichen planus of the back.

Pseudopelade of brocq

The patients include 1 male and 2 females. There were multiple skin colored irregular and atrophic patches of scarring alopecia of variable sizes. Two

patients had classical foot print in snow appearance. There was no history of any inflammation on the affected area. The lesions were asymptomatic.

Acne keloidalis nuchae

Both patients were male . Both presented with cicatricial plaque with painful pustules.

Keratosis spinulosa declavans

Plaques on the scalp was cicatrized with few pustules. The patient had no other features of KSD.

Secondary cicatricial alopecia

Ten (20%) patients had cicatricial alopecia due to secondary insult to the hair follicle. Trauma and naevus sebaceous were the most common cause 2(2%) followed by 1% each of post Radiotherapy, lipoid proteinosis, Gunthers , Trichilemmal cyst , Seborrheic keratosis and En coup de sabre.

TABLE 9.A. DERMASCOPIC FEATURES OF CICATRICIAL ALOPECIA

	Number of Patients	Percentage
Black dot	0	0
Classic white dot	10	20
Absent follicular opening	50	100
Yellow dot	1	2
Pustules	3	6
Hyperkeratosis	13	26
Follicular plugging	4	8
Peripilar cast	17	34
Thick arborising blood vessel	10	20
Linear blood vessel	4	8
Brown discolouration	13	26
Erythema	15	30
Scaling	23	46
Atrophy	41	82
Cicatricial white patch	30	60
Blue grey dot	9	18
Crypts and yellow globules	2	4

TABLE 9.B. GRAPHICAL REPRESENTATION OF DERMASCOPIC FEATURES OF CICATRICIAL ALOPECIA

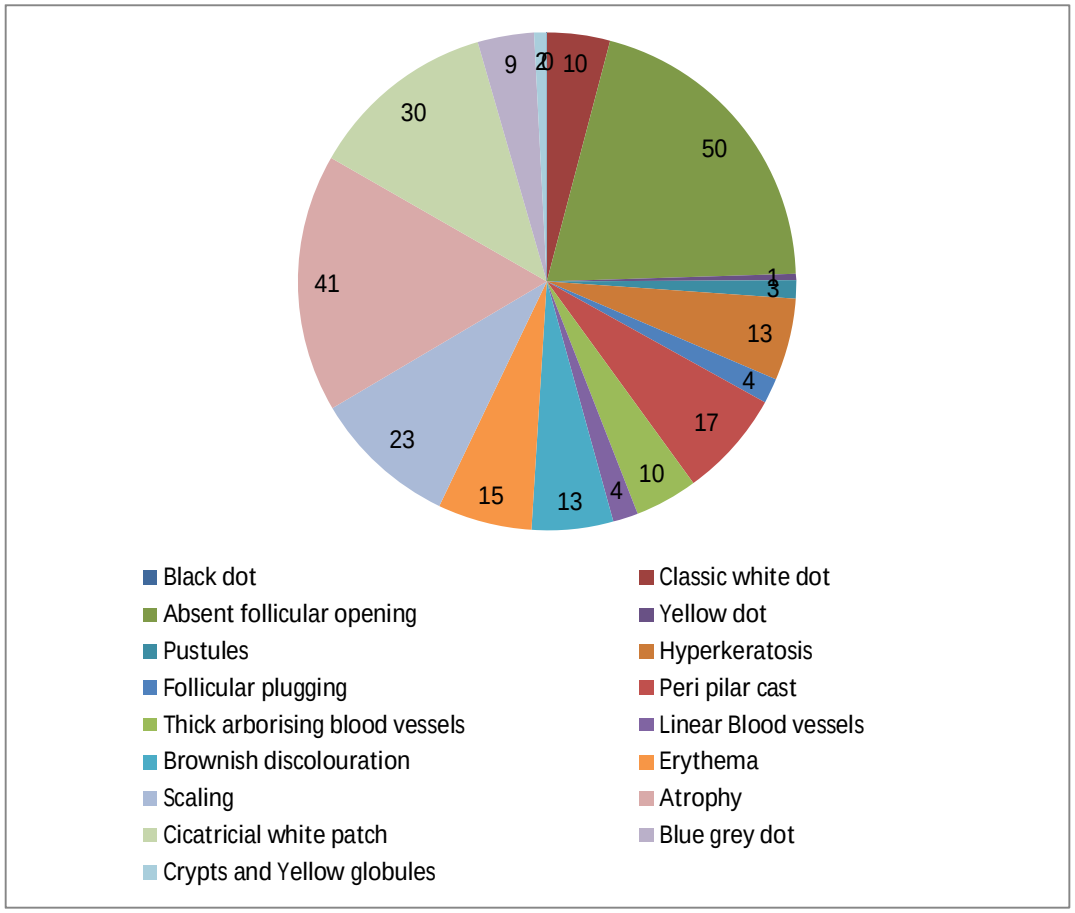


Table 9 shows dermascopic features of cicatricial alopecia. Dermascopy showed an absence of follicular ostia in all patients. Epidermal atrophy was seen in 82% of patients. Linear and thick arborising blood were predominantly noted in patients of DLE. Peripilar cast and blue grey dots were characteristically seen in patients with LPP. Naevus sebaceous was characterized by crypts and yellow globules. Significant differences in frequency of dermascopic signs were noted in various cases.

TABLE 10.A.DERMASCOPY AND PRIMARY CICATRICIAL ALOPECIA

	DLE N=13	**LPP N=15**	**POB N=3**	**KSD N=1**	**AKN N=2**	**Non. Specific CA N=4**	**P VALUE**
Black dot	0	0	0	0	0	0	-
Classic white dot	0	3	1	1	0	2	0.8
Absent follicular opening	13	15	3	1	2	4	**0.012**
Yellow dot	0	0	0	0	0	1	0.7
Pustules	0	0	0	1	0	0	**.001**
Hyperkeratosis	5	6	0	0	0	1	0.2
Follicular plugging	0	1	0	0	2	0	**.002**
Peripilar cast	0	14	0	0	0	0	**.001**
Thick arborising blood vessel	10	0	0	0	0	0	**.003**
Linear blood vessel	4	0	0	0	0	0	0.7
Brown discoloration	3	6	0	0	0	2	.446
Erythema	13	0	0	0	2	0	**.001**
Scaling	10	8	0	0	0	1	.092
Atrophy	14	`11	2	1	2	3	**0.025**
Cicatricial white patch	12	8	2	0	0	1	**0.055**
Blue grey dot	0	8	0	0	0	0	.153

Table 10 shows the different dermascopic features in different causes of primary cicatricial alopecia. Absence of follicular opening , thick arborising blood vessel, pustules show a p value less than .01 which signifies they are highly significant. Atrophy and cicatricial white patch have a p value > .01 but < .05 which implies they are significant .

TABLE 11.A.HISTOLOGICAL FEATURES OF CICATRICIAL ALOPECIA

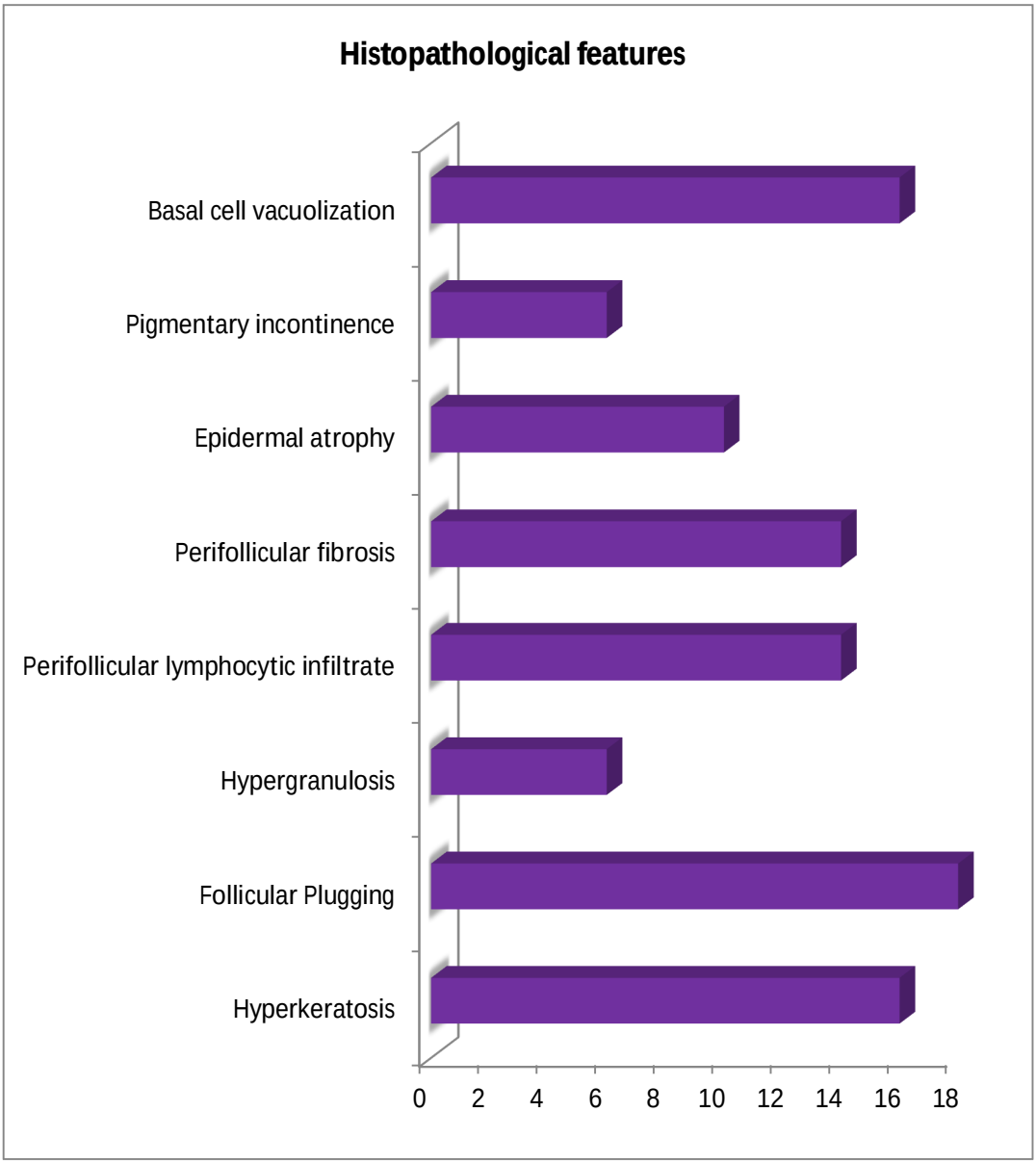


TABLE 11.B. GRAPHICAL REPRESENTATION OF HISTOPATHOLOGIC CHARACTERISTICS OF CICATRICIAL ALOPECIA

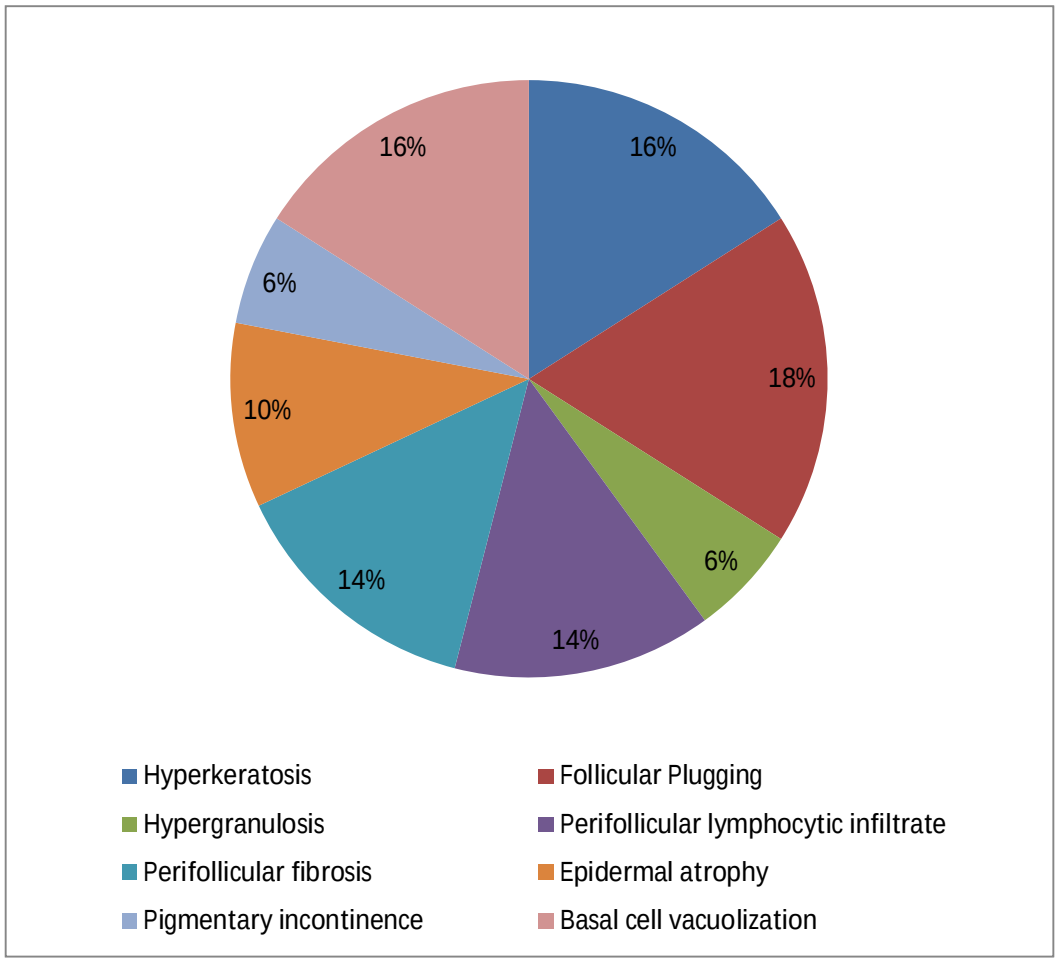


Table 11 shows that the commonest histopathological feature of cicatricial alopecia was follicular plugging (18%) followed by basal cell vacuolization and hyperkeratosis (16%) each. Other features were 14% of perifollicular fibrosis and perifollicular lymphocytic infiltrate. Epidermal atrophy (10%),hypergranulosis and pigmentary incontinence 6% were noted.

TABLE 12. HISTOPATHOLOGICAL FEATURES IN PRIMARY CICATRICIAL ALOPECIA.

	DLE N=13	LPP N=15	POB N=3	KSD N=1	AKN N=2	P VALUE
Epidermal atrophy	6	5	3	-	-	.158
Hyperkeratosis	12	14	-	-	-	.607
Follicular Plugging	12	10	-	-	1	.051
Hypergranulosis	-	15	-	-	-	.005
Perifollicularlymphocytic infiltrate	7	5	-	1	2	.765
Basal cell vacuolization	12	15	-	-	-	.532
Pigmentary incontinence	4	14	-	-	-	.055
Atrophied hair follicle	4	5	3	1	2	.03
Dermal sclerosis	-	-	-	-	-	-
Absent sebaceous gland and hair follicle	4	-	1	-	2	.134

Table 12 shows the histopathological features in various types of Primary cicatricial alopecia. There are significant differences between each type of cicatricial alopecia and these differences are found to be significant with the help of p value derived.

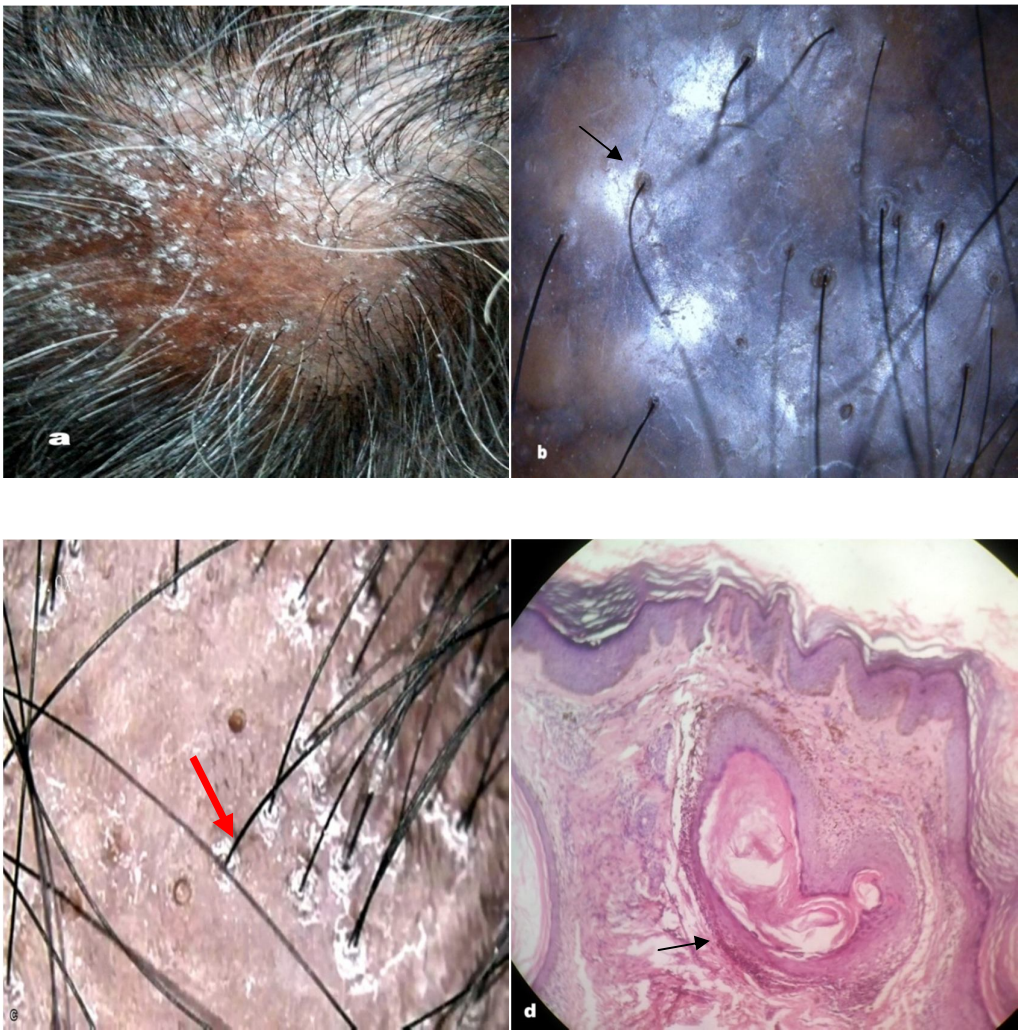


Image 1:Lichenplano pilaris

a – Atrophied patch of LPP showing perifollicular hyperkeratosis

b&c – Dermascopy showing Blue grey dots and Peripilar cast

d – Histopathology showing keratotic plugging, hypergranulosis, hair follicle basal cell degeneration with pigmentary incontinence.

Image 2: Graham Little Picardi Syndrome

a – cicatricial alopecia of scalp, b – follicular papules in back, c – non scarring alopecia of axilla, d – dermascopy showing follicular plugging e – HPE showing thinning of epidermis , hair follicle basal layer degeneration and pigmentary incontinence.

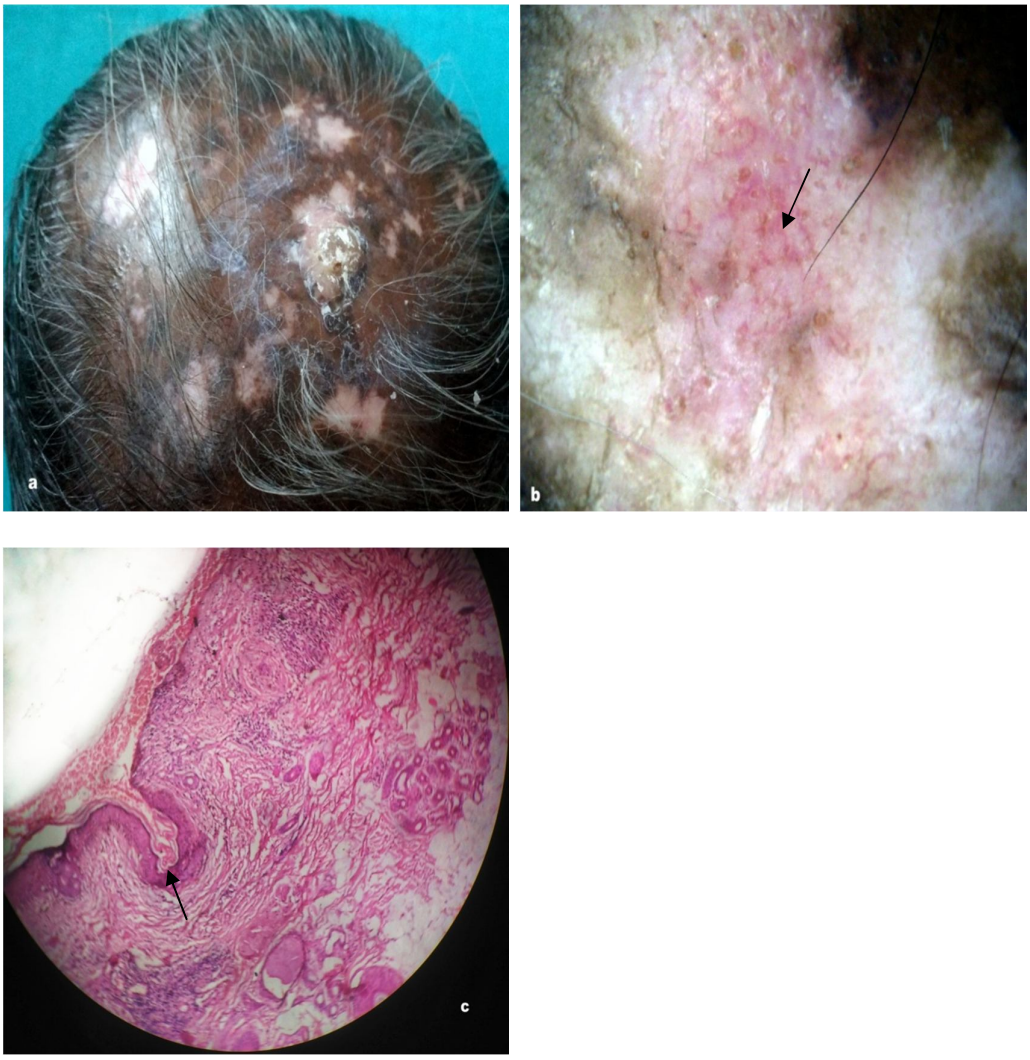


Image 3 : Discoid Lupus Erythematoses

a – DLE plaque with central atrophied depigmentation and peripheral hyperpigmentation, with adherent scales and telengiectasia.

b – Dermascopy shows absent follicular opening, arborising and linear blood vessels

c – HPE shows keratotic plugging, epidermal atrophy, focal basal cell degeneration and plenty of lymphocytic infiltrate.

Image 4 : Pseudo pelade of Brocq

a- Foot print in snow appearance

b – Dermascopy showing absent follicular opening

c – HPE showing epidermal atrophy , atrophied hair follicles and absence of inflammatory infiltrate.

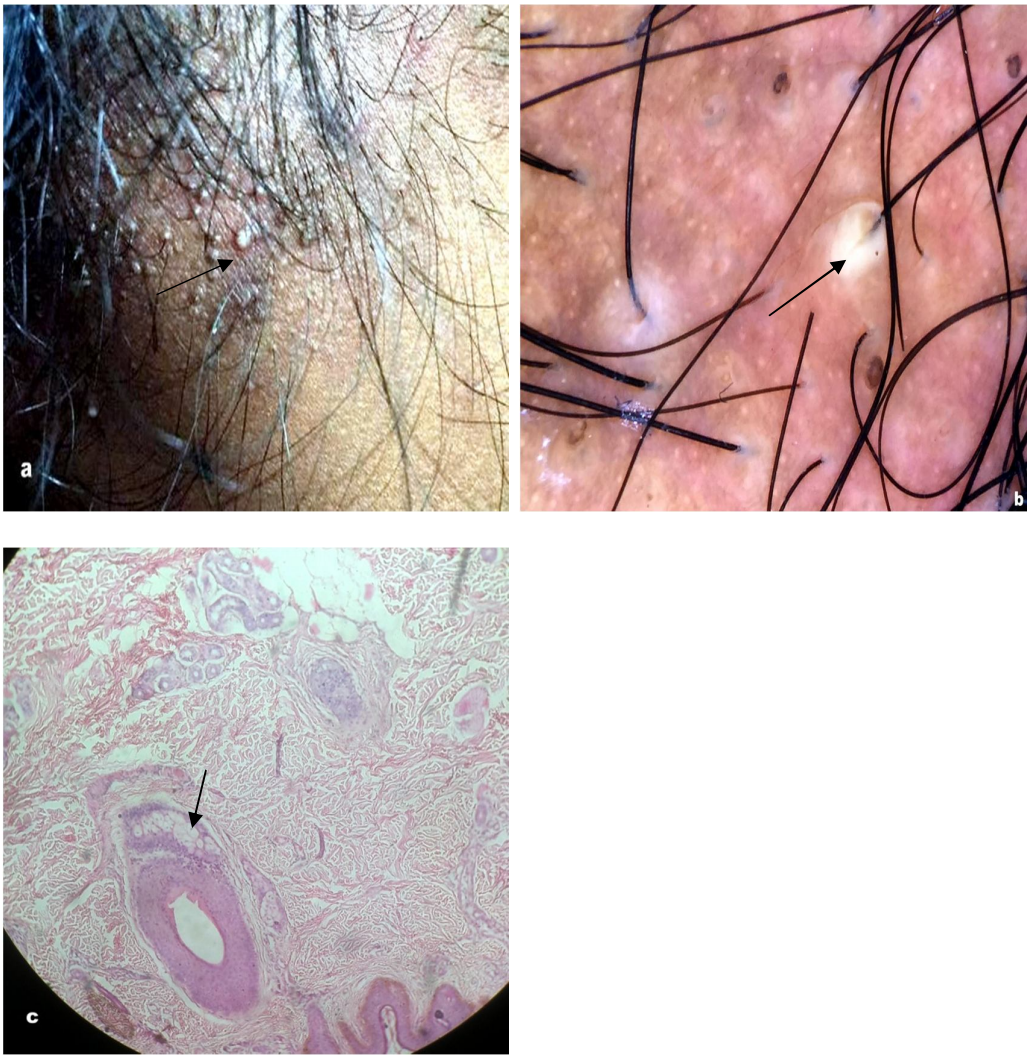


Image 5: Keratosis spinulosa declavans

a- cicatricial patch with pustules

b – Dermascopy showing white dot and pustules

c – HPE showing empty hair follicle with sparse peri follicular lymphocytic infiltrate and dermal edema

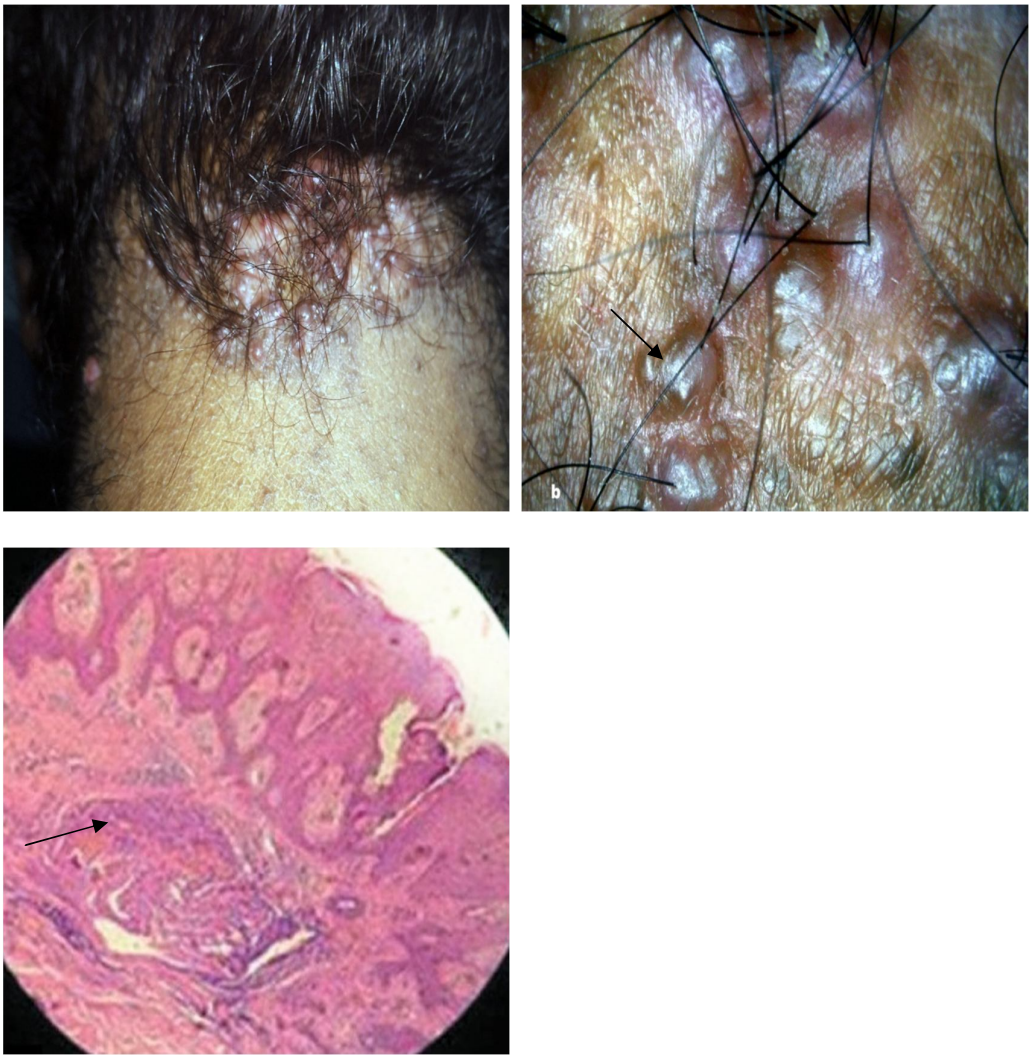


Image 6 : Acne keloidalis nuchae

a – papules and nodules in occiput and nape of neck

b – Dermascopy showing atrophy, erythematous papule , and broken hair.

c - HPE showing acanthosis, plenty of lymphocytic infiltrate around epidermal appendages

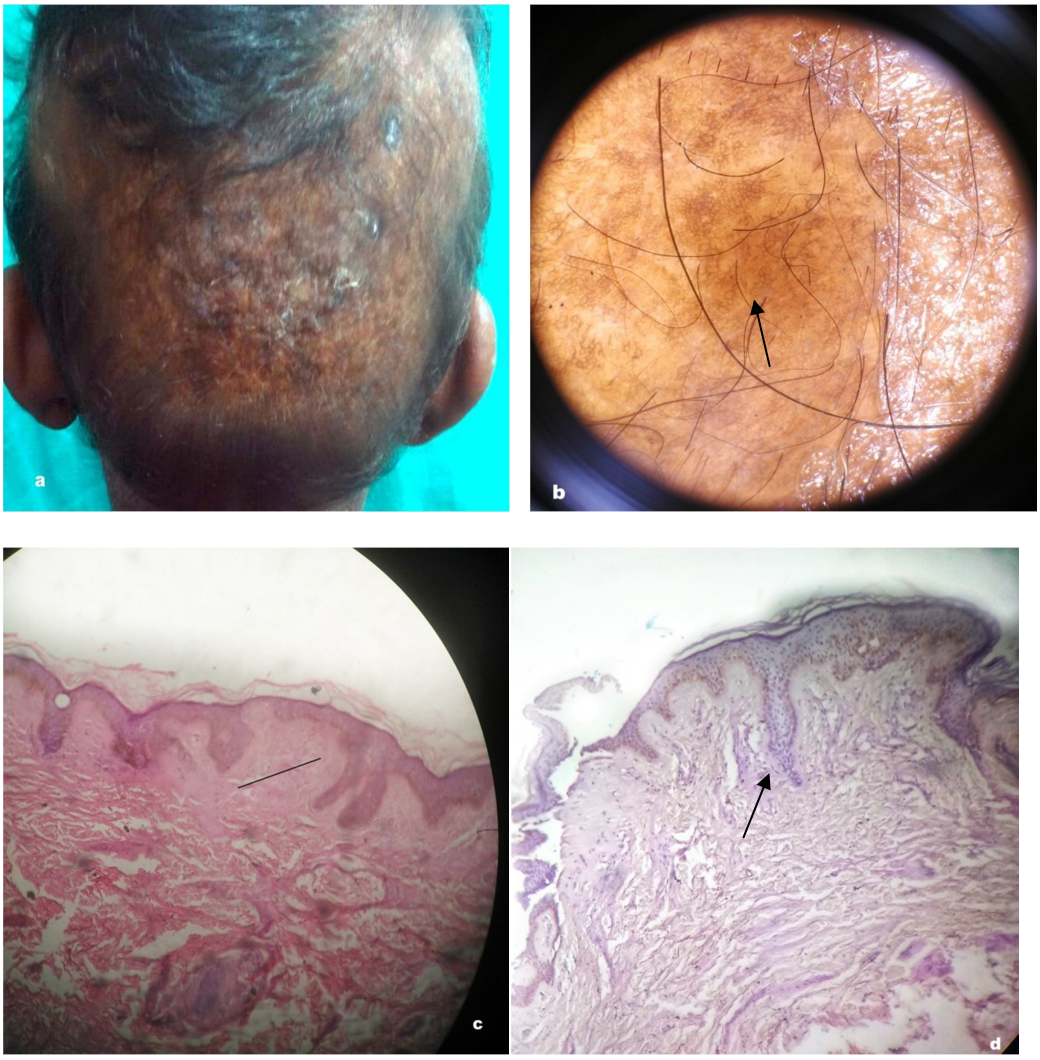


Image 7 : Lipoid Proteinosis

a-atrophied waxy papules seen in occiput

b – Dermascopy showing absent follicular opening, brownish discoloration, atrophy and cicatricial white patch.

c- HPE showing atrophy of epidermis, pink hyaline material and atrophied hair follicle.

d- PAS staining showing PAS positive substance at basement membrane

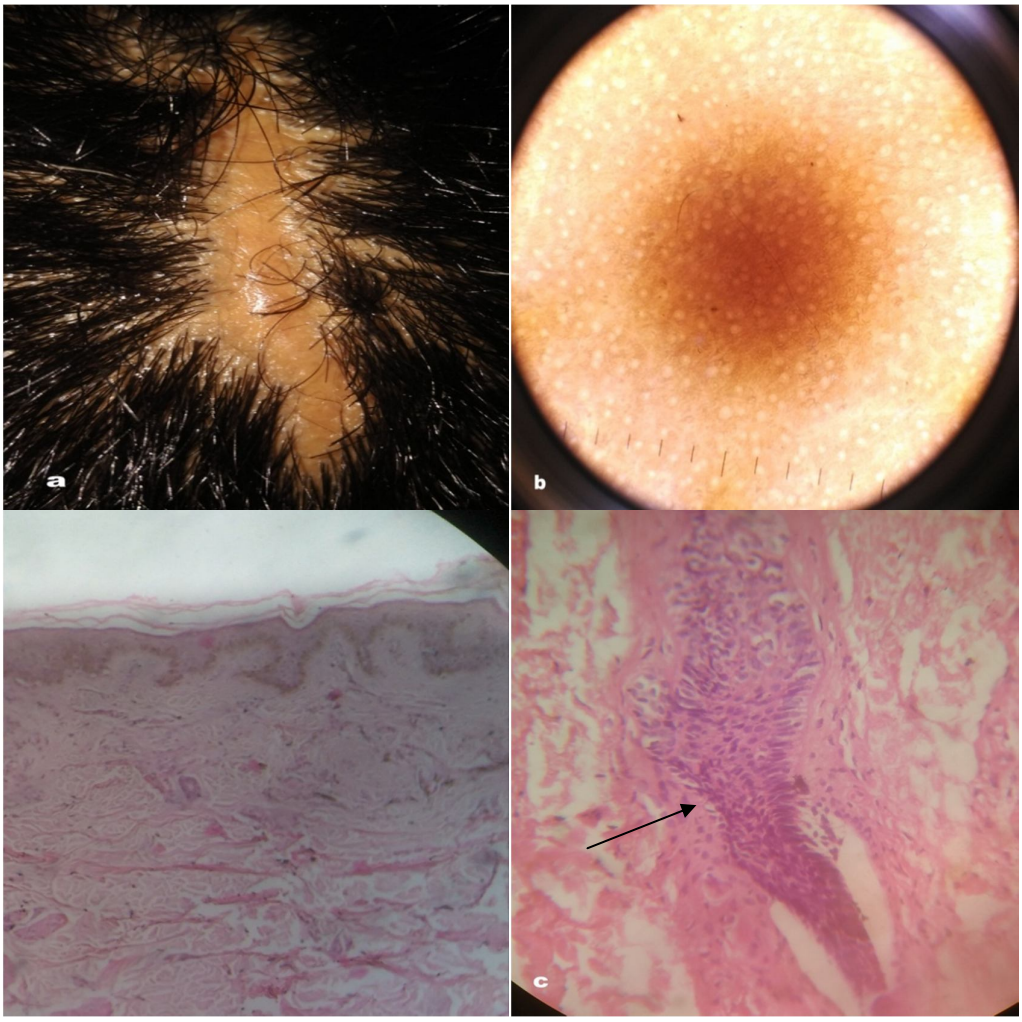


Image 8 – Post traumatic cicatricial alopecia

A – atrophied patch

B – Dermascopy – white dot, absent follicular ostia

C&D –sparse hair follicles seen (d)and High power view showing atrophied hair follicle (c)

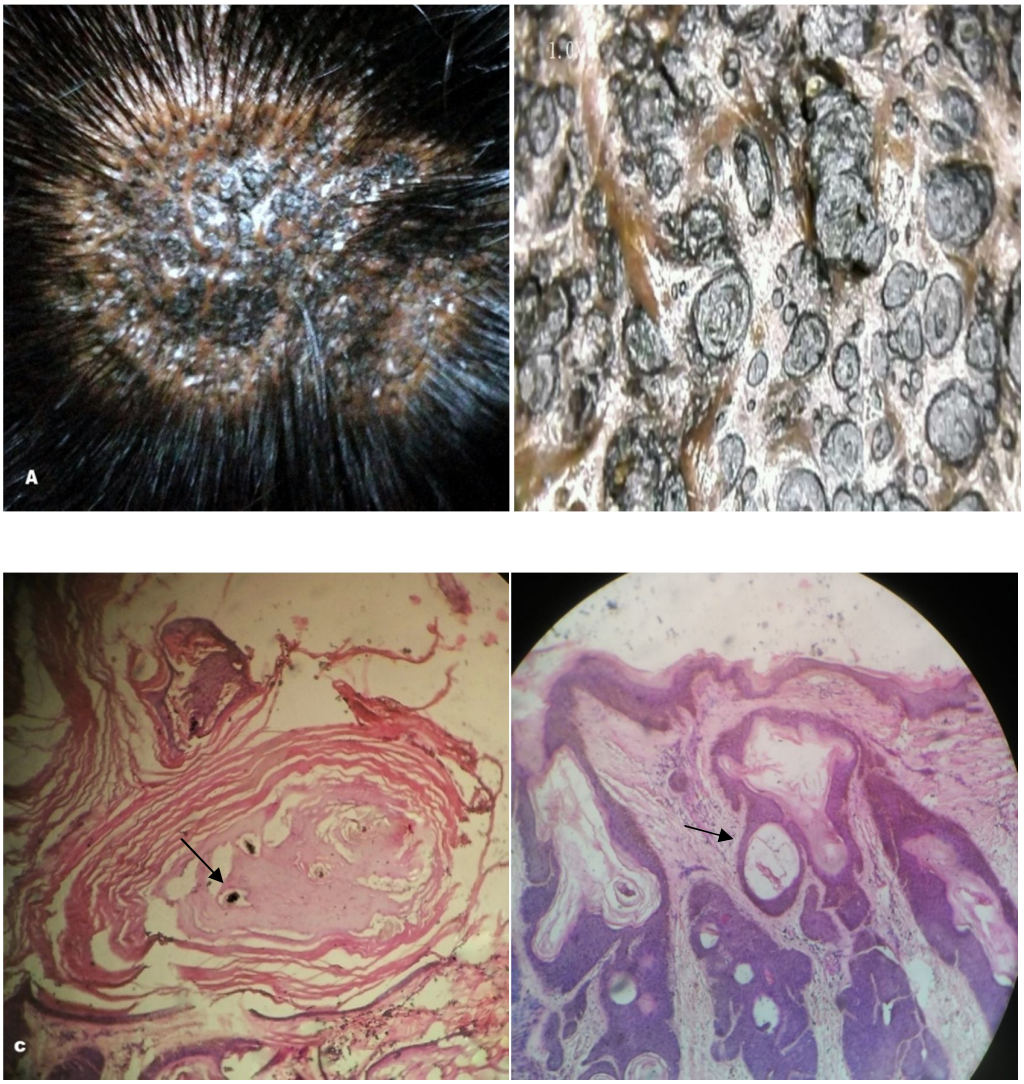


Image 9 : Trichilemmal cyst

A – well defined cicatricial patch with keratotic follicular plugs and nodules

B – Dermascopy shows hyperkeratosis , follicular plugging

C&D – HPE showing hair follicle forming cyst. Hair cyst lined by squamous epithelium with abrupt keratinisation. Foci of calcification seen in Von Kossa stain(c).

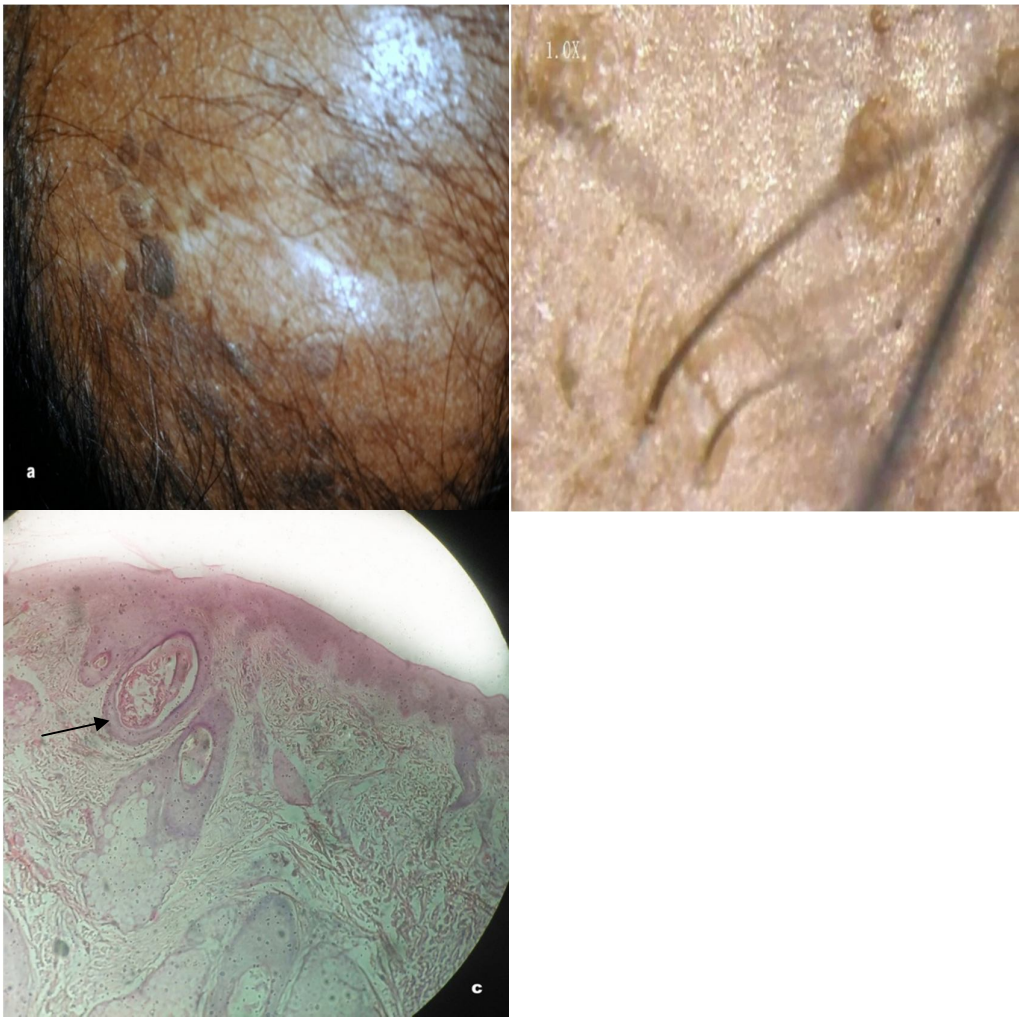


Image 10 – Radiation induced cicatricial alopecia

A – atrophied indurated patch

B – Dermascopy showing absent follicular ostia

C – HPE showing thinning of epidermis and atrophied hair follicle

Image 11– Seborrheic keratosis – Hyperkeratotic Type

A – well defined hyperkeratotic plaque with ridges and crypt

B – Dermascopy showing irregular crypts with fissures and ridges and grey globules

C – massive hyperkeratosis and papillomatosis. Digitate upward extensions resembling church spire pattern

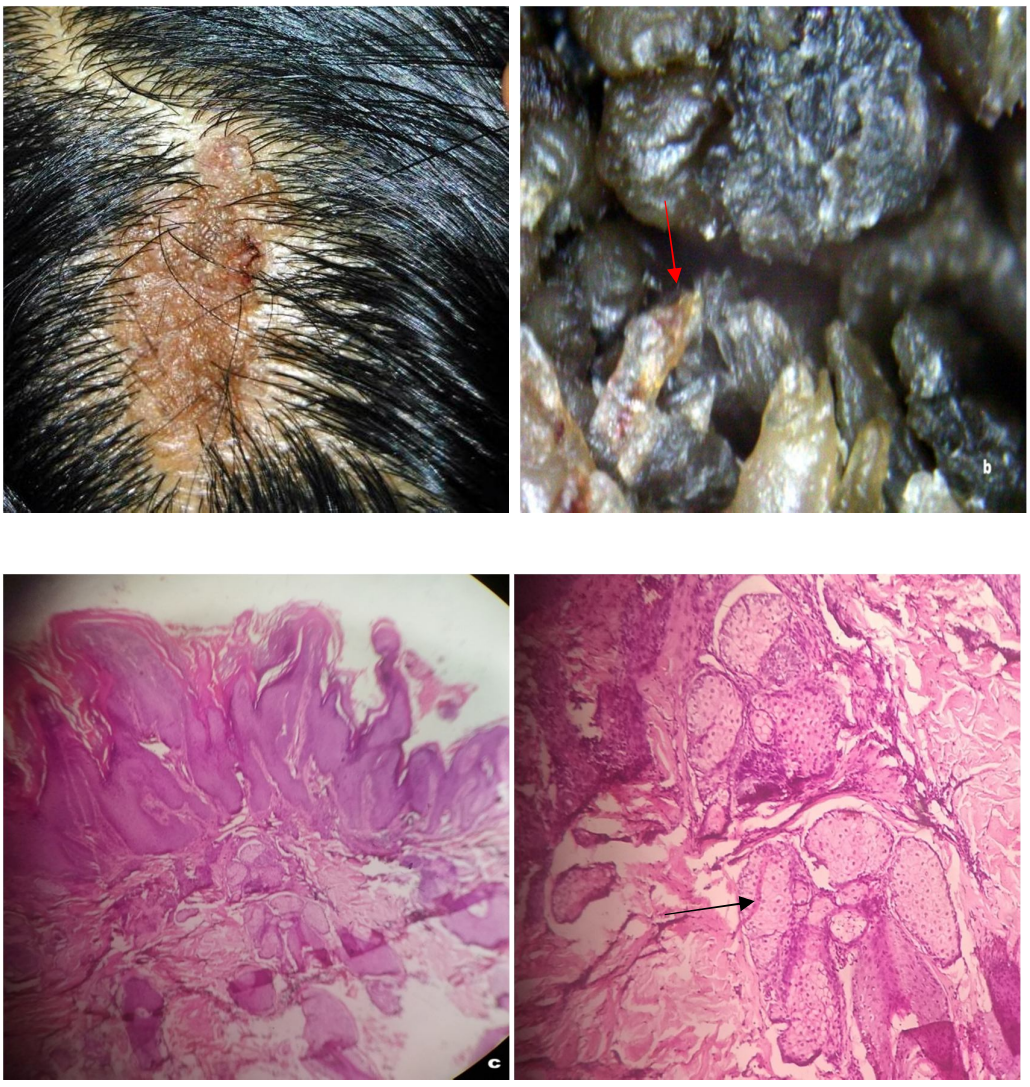


Image 12 – Naevus sebaceous

A – well defined smooth yellowish greasy plaque

B – Dermascopy showing crypts, yellow globules

C – HPE showing epidermal hyperkeratosis, papillomatosis and multiple sebaceous glands seen in high power (d)

Image 13 – Gunthers disease

a- Cicatricial alopecia

b- Dermascopy showing Crusts and hyperkeratosis

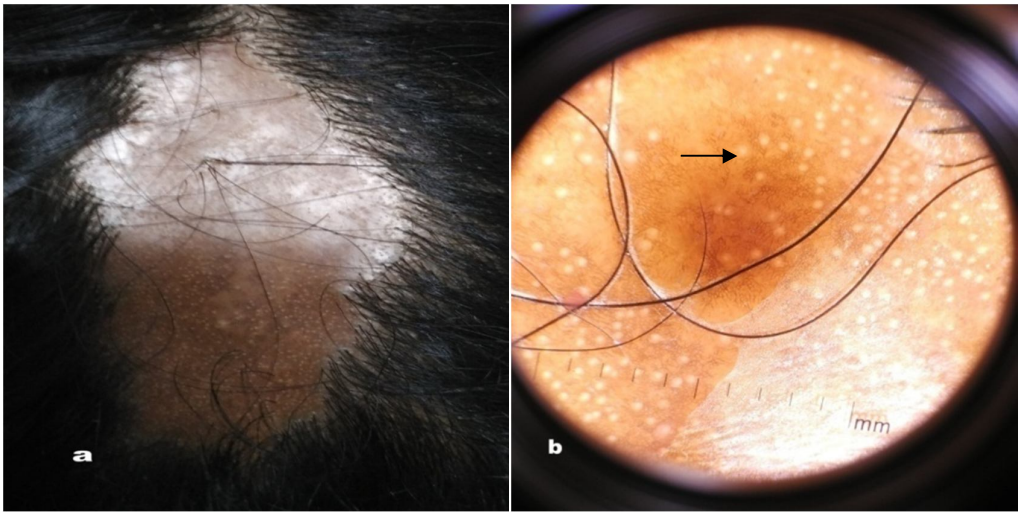
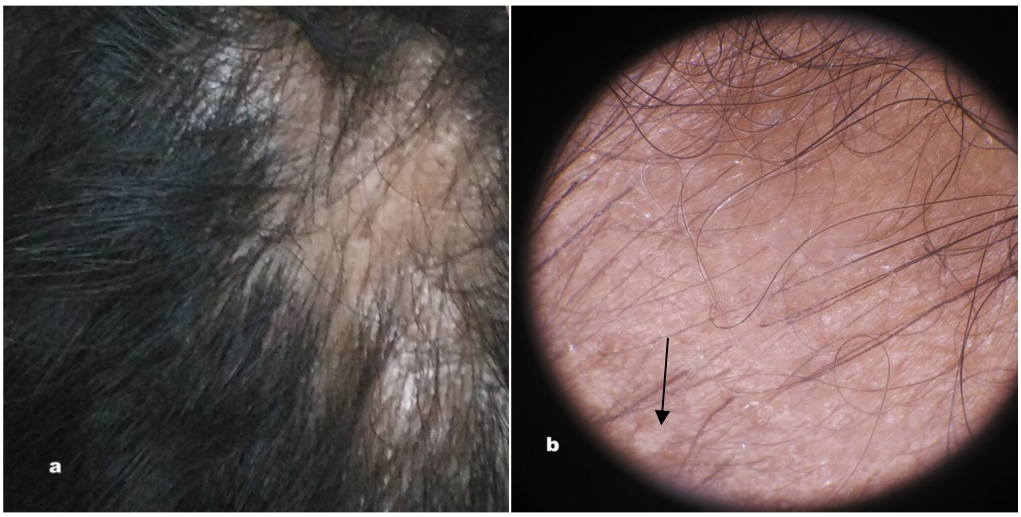


Image 14: Non specific cicatricial alopecia

A – atrophy , absent follicular ostia

B – white dot, sparse follicular opening and few terminal hairs.



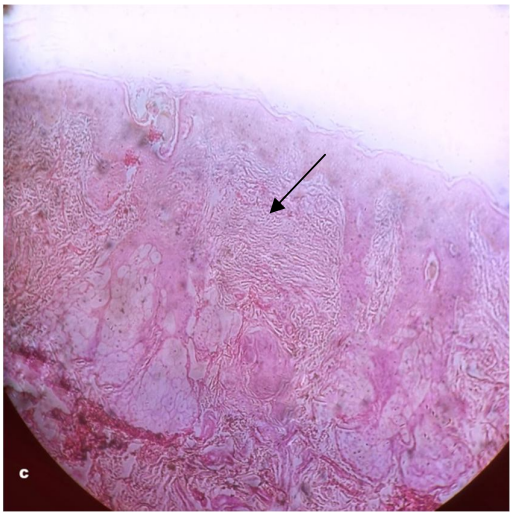


Image 15 – En coup de sabre

A – atrophied patch with induration

B – Dermascopy – cicatricial white patch

C – flaky hyperkeratosis,follicular plugging,atrophy of dermis,hypertrophic collagen in dermis and sub cutis

Discussion

DISCUSSION

Primary cicatricial alopecia are a group of disorders which can result in permanent hair loss. The follicles are replaced by fibrosis.[66]

In Primary cicatricial alopecia the hair follicle is the main target of an inflammatory process. A study done by Whiting et al has reported that the condition comprised 7.3% of all cases of hair loss[4.] UK survey by Griffin et al revealed about 9.6 new cases of primary cicatricial alopecia diagnosed per year.[67]

There are very limited studies on cicatricial alopecias from India.

In this study total of fifty patients were studied . 80% of patients constituted Primary cicatricial alopecia and 20% of cases were secondary cicatricial alopecia with a ratio of 4: 1. This is comparable with study made by Whiting et al[4] , a large retrospective study over 10 years in which the ratio of Primary to secondary cicatricial alopecia was 4:1.

In my study 31 – 40 yrs constituted majority (30%) of cicatricial alopecia patients as shown in table 1. The minimum age was 4 and maximum 68 with a mean 35.46 and standard deviation of 15.032. The disease onset is found to be gradual in 96% of patients (table 3) . The minimum disease duration was 8 months and maximum 20 years with mean of 3.96 years and standard deviation of 4.5 .

Study by Qi et al[74] from China constituted age range of 5 to 74 years with mean age of 31.7 years. Duration of disease ranged from 1 month to 10 years with an average of 1.6 years.

Minor variations in these studies can be due to ethnic and demographic variations. This highlights the indolent and progressive course of cicatricial alopecia and hence early diagnosis aids in halting the disease progress.

In my study there were 56% of females and 44% of males in the ratio 1.3: 1which signifies predominant female patients being affected by cicatricial alopecia. Study by Thakur et al[75] in North East india had Female to male ratio 2:1.

This shows that females are affected more than males , though there may be a slight difference in the ratio which can be attributed to the geographical variations.

Incidences of various types of Primary cicatricial alopecia reported in Qi et al[74] , Thakur et al[75] and Whiting et al[4] compared to our study is summarized as follows;

	Our Study n= 50	**Qi et al n = 59**	**Thakur et al n= 24**	**Whiting et al n=358**
DLE	13(26.0)	13(22.0)	10(41.7)	31(22.0)
LPP	15(30.0)	2(3.4)	5(20.8)	36(10.1)
POB	3(6)	9(15.3)	2(8.3)	116(32.4)
AKN	2(4)	-	-	-
GLP	1(2)	-	-	-

In my study Lichen Plano Pilaris was the most common etiology followed by DLE. This is in discordance with most other studies wherein DLE is the most common cause of cicatricial alopecia. This can be attributed to the decreased incidence of Lupus erythematoses in coloured races.

In my study there was paucity of alopecia mucinosa and neutrophilic primary cicatricial alopecia like Folliculitis declavans and Dissecting cellulitis of scalp. This can be attributed to the difference in time frame of survey ,limited number of cases studied and territorial variations.

Very few studies on Secondary cicatricial alopecia have been published in India . Trauma, radiation , scalp tumors , lipoid proteinosis and Gunthers were the few causes of secondary cicatricial alopecia seen in my study. There are wide number of causes of secondary cicatricial alopecia and in the limited timeframe of this study its beyond its scope to find a significant number of patients presenting with cicatricial alopecia secondary to other causes.

Dermascopy is a noninvasive tool which provides clues for diagnosis and biopsy localization.[69]

Dermatoscopy of scarring alopecia is characterized by decreased hair density and loss of follicular openings in almost 100% cases.[38]

Lichen Plano Pilaris is characterized by peri pilar cast and blue grey discolouration.(Fig 1.b) . Graham Little syndrome is an variant of lichen plano pilaris with cicatricial alopecia of scalp and non cicatricial alopecia of other site with follicular lichen planus.(fig 2). It shares the dermascopic and histopathological features of LPP. These findings of dermascopy are positively correlated with Lichen Plano Pilaris ($p < 0.01$)

Arborising and linear blood vessels were noted in most of the cases of discoid lupus erythematosus (fig 3.b)of the scalp with a statistical significance of $p < .001$.

The above values can be correlated with that of Thakur et al[75] and Qi et al[74] in which Peripilar cast , arborising and follicular plugging are found to be statistically significant.

The significance of the statistical values derived in our study is compared with that of Qi et al[74] and Thakur et al[75] and summarized as follows

	Our study P value	**Qi et al P value**	**Thakur et al P value**
Absent follicular opening	<0.01	<0.01	-
Pustules	<0.01	<0.01	<0.01
Follicular Plugging	<0.01	<0.01	<0.01
Thick arborising blood vessel	<0.01	<0.01	<0.01
erythema	<0.01	-	<0.01
Linear blood vessel	>0.01	>0.01	<0.01
One haired follicle	>0.01	-	<0.01

Pseudopelade of Brocq (PB) is a permanent progressive scarring alopecia characterized by numerous alopecic patches localized only in the scalp, that tend to coalesce into larger, irregular plaques with policyclic borders(fig 4). My patients showed cicatricial white patch and atrophy . No specific dermascopy features have been reported in any previous studies. Thus pseudopelade of brocq is a diagnosis of exclusion.

The classical dermascopic features of acne keloidalis nuchae like tufting of hair follicles , ingrown hairs were not noted in our study due to the paucity in the number of cases studied.

The secondary cicatricial alopecias shared the common feature of absent follicular ostia . Naevus sebaceous and seborrheic keratosis had presence of crypts, yellow globules , cerebriform surface and fat fingers. The significance of this could not be concluded due to the limited number of these patients in our study.

Thus dermascopy may aid as a screening tool which is non invasive. A presumptive diagnosis can be made with dermascopy which can be further confirmed with biopsy.

Scalp biopsies are crucial for the accurate diagnosis and differentiating of cicatricial alopecia.[70] Based upon the histopathological picture, the Cicatricial alopecia are divided mainly into lymphocyte-mediated and neutophil-mediated primary cicatricial alopecia and mixed CA [71.]

In my study Lymphocytic Primary cicatricial alopecia constituted 98% and neutrophilic / mixed cicatricial alopecia was only 2 % of the total primary cicatricial alopecia cases. This is not in accordance with previous study in which 20% of Primary cicatricial alopecia studied were neutrophilic mediated. This may be due to the limited time period of the study and may also be attributed to the geographical and ethnic variations.

Lymphocytic mediated alopecias are typified by a lymphocyte rich infiltrate in early stages and with variable but limited fibrosis concentrated in the perifollicular adventitial unit in later stages. In contrast, in neutrophilic group there is a neutrophil-rich infiltrate in early disease, a mixed infiltrate in later disease, and marked scarring that extends beyond the peri-follicular dermis and extensively involves the reticular (inter-follicular) dermis.[72]

In mixed CA group inflammatory infiltrate is mixed lymphocytic and neutrophilic.

Hypergranulosis , basal cell vacuolization and pigmentary incontinence were seen in most of the cases of Lichen Plano Pilaris.

Patches of Discoid lupus erythematoses showed keratotic follicular plugging , focal basal cell vacuolization and perifollicular lymphocytic infiltrate.

Atrophy of epidermis and decrease in hair follicles were noted in cases of pseudo pelade of brocq.

Acne keloidalis nuchae showed presence of flaky hyperkeratosis, plenty of lymphocytic infiltrate around epidermal appendages.

Keratosis folliculitis spinulosa declavans was characterized by perifollicular lymphocytic infiltrate with few atrophied follicles. The patient did not have any skin manifestations or loss of eyebrows. This can be explained that the disease is more severe in males and females have a milder course.

Secondary cicatricial alopecia shared common feature of paucity of hair follicles and atrophy of epidermis. Lipoid proteinosis patient had deposition of hyaline material between rete ridges which was confirmed on PAS staining. Both the patients of naevus sebaceous had epidermal hyperplasia , papillomatosis and multiple mature sebaceous glands. Seborrheic keratosis had marked acanthosis and papillomatosis . Abrupt keratinization and foci of calcification were noted in Trichilemmal cyst.

Amongst this follicular atrophy, hypergranulosis and Keratotic follicular plugging, pigmentary in continence are statistically significant ($p < 0.05$).

Study by Qi et al and Thakur et al has shown similar statistical significance ($p<0.05$) in keratotic follicular plugging and follicular atrophy. In their study basal cell vacuolar , neutrophilic folliculitis , perifollicular lymphocytosis , parakeratosis were also found to be significant($p<0.05$) . Such variations can be attributed to the fact that there were variations in the number of specific cicatricial alopecia between these studies and ours.

Four of my patients who had clinically cicatrized patch and absence of follicular ostia in dermascopy. In histopathology other than thinned out epidermis and follicular atrophy other features were inconclusive. They were grouped under non specific cicatricial alopecia.

Moure ERD et al has quoted 13 cases of non specific cicatricial alopecia amongst 38 patients they studied. Literature shows such cases may be burnt out stages of any specific cause or may be an early presentation of LichenPlano Pilaris. These could not be concluded in this study due to the short time frame .

Limitations of the study

LIMITATIONS OF STUDY

A limitation of this study is the small number of patients in each type of PCA. Patients with cicatricial alopecia report late to the hospital when the disease has already progressed and reached a burnt out stage. Patients with cicatricial alopecia during the early phase may be misdiagnosed as non scarring alopecia or alopecia areata. Histopathology remains standard tool for confirmatory diagnosis. This imposes a drawback as it is an invasive procedure.

Conclusion

CONCLUSION

1. Primary cicatricial alopecias are uncommon disorders and increasing the awareness is necessary to improve the management.
2. Dermascopy may be reliable in differentiating DLE and LPP from other primary cicatricial alopecias.
3. Histopathology is the standard tool to find the cause of primary cicatricial alopecia which may not have characteristic features clinically and in dermascopy.
4. Histopathology is the final confirmatory diagnostic tool.
5. Few cases of Primary cicatricial alopecia may be inconclusive in histopathology as well, which are categorized as non specific cicatricial alopecia . These patients need to be followed up closely as they may progress to a specific form in latter stages.
6. If not diagnosed in early phase there will be inevitable loss of hair follicle and scarring alopecia ensues.
7. The treatment of cicatricial alopecias are not promising , hence prompt diagnosis at an early stage remains crucial for preventing permanent scarring.

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Annexures

ABBREVIATIONS

CA	–	cicatricial alopecia
DLE	–	Discoid Lupus Erythematoses
SLE	–	Systemic Lupus Erythematoses
LPP	–	Lichen Plano Pilaris
POB	–	Pseudopelade of Brocq
KSD	–	Keratosis spinulosa declavans
AKN	–	Acne keloidalis nuchae
PCA	–	Primary cicatricial alopecia
I/L	–	Intralesional
ORS	–	outer root sheath
PPAR	–	Peroxiosome proliferated activated receptor
HIV	–	Human immune deficiency virus
DIF	–	Direct Immuno fluorescence
BMZ	–	basement membrane zone
CCLE	–	chronic cutaneous lupus erythematoses
SK	–	Seborrheic Keratosis

CICATRICIAL ALOPECIA - CLINICAL , DERMASCOPY AND HISTOPATHOLOGICAL STUDY - MASTER SHEET

Sl No	NAME	AGE	SEX	OP No	HISTORY OF HAIR LOSS														CLINICAL EXAMINATION							DERMASCOPY												HISTOPATHOLOGY
																			SCALP							FOLLICULAR								PERIFOLLICULAR							
					Duration	Gradual /Acute	PATCHY /DIFFUSE	Thinning /Shedding	Recession of hair Line	Pain/Burning	Itching	Photosensitivity	Scaling	Discharge	Scaring	Regrowth of Hair	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY	SITE	Number	PATCHY/ DIFFUSE	Hair pull Test	Hair Loss in other site	Skin lesions	Oral / Genital mucosa	Palms,soles &nails	Black Dot	Yellow Dot	Classic white dot	Absent follicular opening	Pustules	Hyperkeratosis	Peripilar cast	FOLLICULAR	Thick arborising BV	Linear BV	Brown	Erythema	Scaling	Atrophy	Cicatricial white	blue grey dot	others	
1	Alagraj	52	M	221197	2 M	G	P	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V,F,P	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	Consistent with Lichen plano pilaris
2	Ragunath	32	M	221187	6 M	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V,F,P,O	MULTIPLE	P	N	Nil	Lichenplanus pigmentosus	N	N	N	N	N	Y	N	N	N	N	N	N	Y	Y	Y	Y	N	Consistent with Lichen plano pilaris with Lichenplanus pigmentosus
3	Rajkamal	28	M	221176	1 Y	G	P	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	P,T	MULTIPLE	P	N	Nil	Lichenplanus pigmentosus	LP	N	N	N	N	Y	N	N	N	N	N	N	Y	Y	Y	N	N	Consistent with Lichen plano pilaris with Lichenplanus pigmentosus
4	Chandra	68	F	230094	6M	G	P	Y	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V,P	MULTIPLE	P	N	Axilla B/L	Lichen Planus	N	N	N	N	N	Y	N	Y	Y	N	N	N	Y	N	Y	N	Y	N	N	Consistent with Grahamlittle syndrome
5	Vijaya	46	F	221199	6 M	G	D	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V,F,P	MULTIPLE	D	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	N	N	N	N	Y	Y	Y	Y	N	Consistent with LPP
6	Punniyakodi	40	F	2E+05	2 Y	G	P	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	N	Y	N	Y	N	Consistent with LPP
7	Krishnamal	45	F	2E+05	2 Y	G	P	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	Consistent with LPP
8	Ranjitham	30	F	220011	8 M	G	D	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V,F,T,P,O	MULTIPLE	D	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	N	N	Y	N	Y	Y	Y	Y	N	Consistent with LPP
9	Anandi	50	F	221197	6 M	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V,O	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	Y	N	N	N	N	Y	Y	Y	Y	N	Consistent with LPP
10	Balamurugan	27	M	2E+05	6 M	G	P	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	N	Y	Y	Y	N	Consistent with LPP
11	Thilagavathy	48	F	221100	3 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V,O,F,P	MULTIPLE	D	N	Nil	Nil	N	N	N	N	Y	Y	N	N	Y	N	N	N	Y	N	N	Y	Y	N	N	Consistent with LPP
12	Malathy	40	F	2E+05	2 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	Y	Y	N	N	Y	N	Y	N	N	Y	Y	N	N	Y	N	Consistent with LPP
13	Thiagarajan	45	M	2E+05	9 M	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	P	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	Y	Y	Y	N	N	Consistent with LPP
14	Suresh	42	M	2E+05	6 M	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	Consistent with LPP

Sl. No	NAME	AGE	SEX	OP No	HISTORY OF HAIR LOSS														CLINICAL EXAMINATION							DERMASCOPY															HISTOPATHALOGY
																			SCALP							FOLLICULAR								PERIFOLLICULAR								
					Duration	Gradual /Acute	PATCHY /DIFFUSE	Thinning /Shedding	Recession of hair Line	Pain/Burning	Itching	Photosensitivity	Scaling	Discharge	Scaring	Regrowth of Hair	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY	SITE	Number	PATCHY/ DIFFUSE	Hair pull Test	Hair Loss in other site	Skin lesions	Oral / Genital mucosa	Palms,soles &nails	Black Dot	Yellow Dot	Classic white dot	Absent follicular opening	Pustules	Hyperkeratosis	Peripilar cast	FOLLICULAR	Thick arborising BV	Linear BV	Brown	Erythema	Scaling	Atrophy	Cicatricial white	blue grey dot	others	
15	Ganesh	28	M	2E+05	6 M	G	P	N	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	Y	Y	N	N	Y	N	N	N	N	N	Y	N	N	N	Consistent with LPP
16	Hari	23	M	221871	5 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	T,V,O	MULTIPLE	P	N	Nil	Nil	N	N	N	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	Non specific Cicatricial Alopecia
17	Malarkodi	28	F	215106	5 Y	G	P	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Non specific Cicatricial Alopecia
18	BoopSingh	27	M	221190	1 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V,P	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	Y	N	N	N	Y	N	Y	N	N	N	N	Non specific Cicatricial Alopecia
19	Krishnamal	45	F	287345	2 Y	G	P	Y	N	N	Y	N	N	N	Y	N	NIL	NIL	NIL	V,P	MULTIPLE	P	N	Nil	Nil	N	N	N	N	Y	Y	N	N	N	N	N	N	N	Y	N	N	Y	N	N	Non specific Cicatricial Alopecia
20	Samundeswari	40	F	221011	2 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	Consistent with DLE
21	Dowlath	36	F	221110	2 Y	G	P	N	N	N	Y	Y	N	N	Y	N	NIL	NIL	NIL	V,O,P,F	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	Y	N	Y	Y	Y	Y	Y	N	N	Consistent with DLE
22	Muthulakshmi	55	F	221191	3 Y	G	P	N	N	N	Y	Y	Y	N	Y	N	NIL	NIL	NIL	V,O,P,F	MULTIPLE	P	N	Nil	DLE	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	Y	N	N	Y	Y	Y	Y	N	N	Consistent with DLE
23	Usha	38	F	2E+05	7 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	Consistent with DLE
24	Shanmugam	6	F	221143	10 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	T,F,P,V	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	Y	Y	N	Y	Y	Y	Y	N	N	Consistent with DLE
25	Subramani	51	M	221144	3 Y	G	P	N	N	N	N	Y	N	N	Y	N	NIL	NIL	NIL	P,V,F	MULTIPLE	P	N	Nil	Disseminated DLE	DLE	N	N	N	N	Y	N	N	N	N	N	Y	Y	N	Y	N	Y	Y	N	N	Consistent with Disseminated DLE
26	Geetha	50	F	221398	4 Y	G	P	N	N	N	Y	Y	N	N	Y	N	NIL	NIL	NIL	V,O,P	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	Y	N	N	N	N	Y	N	N	Y	Y	Y	Y	N	N	Consistent with DLE
27	Balakrishnan	42	M	2E+05	3 Y	G	P	N	N	N	Y	Y	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Disseminated DLE	DLE	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	Y	Y	N	N	Consistent with Disseminated DLE
28	Rukmani	40	F	2E+05	10 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	Y	Y	N	N	N	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	N	Consistent with DLE

Sl. No	NAME	AGE	SEX	OP No	HISTORY OF HAIR LOSS														CLINICAL EXAMINATION								DERMASCOPY														HISTOPATHALOGY
					Duration	Gradual /Acute	PATCHY /DIFFUSE	Thinning /Shedding	Recession of hair Line	Pain/Burning	Itching	Photosensitivity	Scaling	Discharge	Scaring	Regrowth of Hair	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY	SCALP				Hair Loss in other site	Skin lesions	Oral / Genital mucosa	Palms,soles &nails	FOLLICULAR								PERIFOLLICULAR								
																				SITE	Number	PATCHY/ DIFFUSE	Hair pull Test					Black Dot	Yellow Dot	Classic white dot	Absent follicular opening	Pustules	Hyperkeratosis	Peripilar cast	FOLLICULAR	Thick arborising BV	Linear BV	Brown	Erythema	Scaling	Atrophy	Cicatricial white	blue grey dot	others	
29	Usha	35	F	221527	2 Y	G	D	N	N	N	N	Y	N	N	Y	N	NIL	NIL	NIL	F,P,O	MULTIPLE	D	N	Nil	Nil	N	N	N	N	Y	Y	N	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	N	Consistent with DLE
30	Lakshmi	25	F	2E+05	2 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F	1	P	N	Nil	Nil	N	N	N	N	Y	Y	N	N	N	N	Y	N	Y	Y	N	Y	Y	N	N	Consistent with DLE
31	Kamatchi	24	F	221138	2 M	G	D	Y	N	N	Y	Y	Y	N	Y	N	Fever,joint	NIL	NIL	F,P,O	MULTIPLE	D	N	Nil	SLE rash	Erosions	N	N	N	N	Y	N	N	N	N	Y	Y	N	N	N	Y	Y	N	N	Consistent with DLE WITH SLE
32	Ganesh	39	M	272205	20 Y	A	P	N	N	N	N	N	N	N	Y	N	Trauma	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Consistent with Post traumatic CA
33	Shubhankar	23	M	235889	10 Y	A	P	N	N	N	N	N	N	N	Y	N	Trauma	NIL	NIL	P	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Consistent with Post traumatic CA
34	Palanivelsamy	67	M	227723	10 Y	G	P	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	P	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	C	Consistent with SK
35	Radha	35	F	23567	5 Y	G	D	N	N	N	N	N	N	N	Y	N	Cranioto	NIL	NIL	F,P,T	MULTIPLE	D	N	Nil	Nil	N	N	N	N	N	Y	N	Y	N	N	N	N	Y	Y	Y	N	N	Consistent with CA
36	Kamaraj	21	M	243567	1 M	G	P	Y	N	Y	Y	Y	Y	Y	Y	N	Gunthers	NIL	NIL	F,P	MULTIPLE	P	N	Nil	scras, erosions, hypertrichiosis	scars	Photo mutilation	N	N	N	Y	N	Y	N	N	N	N	N	N	N	N	Y	Y	N	N	Crust	Consistent with Gunthers disease
37	Sameera Banu	8	Fch	265435	8 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	C,YG	Consistent with Naevus sebaceous
38	Vishnu	17	M	2E+05	17 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	O	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	C,YG	Consistent with Naevus sebaceous
39	Kannan	35	M	239084	6 M	G	P	Y	N	Y	N	N	N	Y	Y	N	NIL	NIL	NIL	O	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	Y	N	N	N	Consistent with AKN
40	Manikannan	36	M	238989	4 Y	G	P	Y	N	Y	N	N	N	Y	Y	N	NIL	NIL	NIL	O	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	Y	N	N	N	Consistent with AKN
41	Nishanti	17	F	230098	7.5 Y	G	P	Y	N	Y	N	N	N	N	Y	N	NIL	NIL	NIL	O	MULTIPLE	P	N	Nil	Nil	N	N	N	N	Y	Y	Y	N	N	N	N	Y	N	N	N	Y	N	N	N	Consistent with KSD
42	Deshnasri	4	Fch	2E+05	4 Y	G	P	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	1	P	N	Nil	Nil	N	N	N	N	N	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	Consistent with Trichilemmal cyst

Sl. No	NAME	AGE	SEX	OP No	HISTORY OF HAIR LOSS															CLINICAL EXAMINATION									DERMASCOPY														HISTOPATHALOGY
																				SCALP									FOLLICULAR								PERIFOLLICULAR								
					Duration	Gradual /Acute	PATCHY /DIFFUSE	Thinning /Shedding	Recession of hair Line	Pain/Burning	Itching	Photosensitivity	Scaling	Discharge	Scaring	Regrowth of Hair	PAST HISTORY	FAMILY HISTORY	PERSONAL HISTORY	SITE	Number	PATCHY/ DIFFUSE	Hair pull Test	Hair Loss in other site	Skin lesions	Oral / Genital mucosa	Palms,soles &nails	Black Dot	Yellow Dot	Classic white dot	Absent follicular opening	Pustules	Hyperkeratosis	Peripilar cast	FOLLICULAR	Thick arborising BV	Linear BV	Brown	Erythema	Scaling	Atrophy	Cicatricial white	blue grey dot	others	
43	Karupaiah	15	M	239908	14 Y	G	D	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	O,P,T	MULTIPLE	D	N	Nil	Waxy papules & scar	scra	Hyperkeratosis	N	N	N	Y	N	N	N	N	N	N	Y	N	N	Y	Y	N	N	Consistent with Lipoid proteinosis
44	Vasantha	58	F	256745	3 Y	G	D	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V,P,O,T	MULTIPLE	D	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	Consistent with POB
45	Krisnan	48	M	287905	2.5 Y	G	P	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F,V,P	MULTIPLE	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Consistent with POB
46	Rajkumari	40	F	287456	2 Y	G	P	Y	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F,V,P,O	MULTIPLE	P	N	Nil	Nil	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	Consistent with POB
47	Danush	32	M	287445	1 Y	G	P	Y	N	N	N	N	Y	N	Y	N	NIL	NIL	NIL	F,P	MULTIPLE	P	N	Nil	NIL	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N	Y	Y	N	N	LPP
48	Vignesh	18	M	2E+05	4 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	F	s	P	N	Nil	Nil	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Consistent with En coup de sabre
49	Kumari	60	F	2E+05	10 Y	G	P	N	N	N	N	N	N	N	Y	N	NIL	NIL	NIL	V	S	P	N	Nil	Nil	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	Y	Y	Y	Y	N	N	Consistent with DLE
50	Kasturi	34	F	3E+05	6 M	G	P	N	N	N	N	y	y	N	Y	N	NIL	NIL	NIL	P	S	P	N	Nil	Nil	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	Y	N	Y	Y	N	N	Consistent with DLE

KEY FOR MASTER CHART

SEX

M	–	Male
F	–	Female

Duration

M	–	months
Y	–	Years
G	–	Gradual
A	–	Acute
P	–	Patchy
D	–	diffuse

History of hair loss

Y	–	Yes
N	–	No

Site

V	–	Vertex
F	–	Frontal
P	–	Parietal
O	–	Occiput
T	–	Temporal

Hair pull test

N	–	Negative
B/L	–	bilateral

Oral and genital mucosa

N	–	Normal
LP	–	Lichen Planus

Palms and soles

N – Normal

Dermascopy

N – No

Y – Yes

Others

C – Crypts

YG – Yellow globules

PROFORMA

Case no: **OP no:**

NAME: **AGE/ SEX:**

ADDRESS: **PH.NO:**

HISTORY:

H/o hair loss – since *

Gradual/sudden

Started in

Spread to

Patchy/diffuse loss of hair	
Gradual thinning of hair:	
Increase shedding of hair:	
Hair breakage:	
Pulling of hair:	
Recession of hair line:	
Pain/itching/burning /scaling/discharge	
Scarring	
Regrowth of hair	
Trauma / burns / radiation / tumors / infection of scalp	
Loss of hair from other body sites:	
Fever, drugs preceding hair loss:	
Irregular periods / hirsutism / OCP:	
Loss of weight:	

Procedure to scalp:	
Skin lesions	

Past history: DM/HT/Tb/chronic illness/similar illness

h/o similar episodes

Family history: similar complaints:

personal history: alcoholic/smoker/tobacco usage

Treatment history:

Clinical examination

SCALP

Site:

Patchy/diffuse:

No.of lesions:

Violaceous papule/Erythema/ Scaling/Pustules/Nodules/abscess/discharge/Crust/Scar

Telengectasia

Mottled hyperpigmentation

Depigmentation with peripheral hyperpigmentation

Broken hairs

Follicular tufting

Induration

Depressed area

tenderness

hair pull test

eyebrows/eye lashes

hair loss in other sites:

Skin Lesions

Lichen Planus

Keratosis pilaris

DLE

Other findings:

NAIL:

ORAL MUCOSA

GENITAL MUCOSA

PALMS AND SOLES

Dermascopy:

Follicular	**YES**	**NO**	**Perifollicular**	**YES**	**NO**
Yellow dot			Thick aborising blood vessel		
Black dot			Linear blood vessel		
Classic white dot			Brown discoloration		
Absent follicular opening			erythema		
Pustules			Scaling perifollicular/interfollicular		
Hyperkeratosis			Atrophy		
Peri pilar cast			Cicatricial white patch		
Predominance of one hair follicle			Blue grey dot,		
Other findings			Yellow crust		
			Other findings		

Histopathological finding:

Investigations:	
CBC	
RFT/LFT	
VCTC	
VDRL	
SCRAPING	
PUS C/S	
ANA	
OTHER INVESTIGATIONS:	

INFORMATION SHEET

TITLE : :" **CLINICAL ,DERMASCOPY AND HISTOPATHOLOGICAL STUDY OF CICATRICIAL ALOPECIA"**

Name of Investigator : Dr.Priyadharsini.J

Name of Participant :

Purpose of Research : The purpose of this study is to analyse the various clinical presentation,dermascopic and histopathological changes in a case of cicatricial alopecia

Study Design : Prospective cohort

Study Procedures : In this study clinical history of the patient with clinical,dermascopic examination and routine blood test will be taken.Scalp biopsy will be done and sent for histopathological study.VCTC,VDRL,pus culture and sensitivity,scraping for tinea capitis ,anti nuclear antibodies and mantoux are done in relevant patients. The patients are treated with topical and systemic drugs according to the diagnosis.

Possible Risks : No risks to the patient

Possible benefits

To patient : The underlying cause for cicatricial alopecia will be detected and patient will be treated accordingly.

To doctor & to other people : The results of the study will help in confirming the role of etiological factors in the causation of the disease with better understanding of the dermascopic and histopathological features and emphasis on early diagnosis and prevention.

Confidentiality of the information obtained from you : The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared

Can you decide to stop participating in the study : Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time

How will your decision to not participate in the study affect you : Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant
Date :
Place :

PATIENT CONSENT FORM

Title of the study: CLINICAL DERMASCOPY AND HISTOPATHOLOGICAL STUDY OF CICATRICIAL ALOPECIA"

Name of the Principal investigator: Dr.Priyadharsini.J.

Name of the Institution: Rajiv Gandhi Government General Hospital,Chennai

Patient's Name :
Patient's Age :
OutPatient No :

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

I understand that my son's/daughter's participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my/ my son's/ daughter's identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study ❑

I hereby give permission to undergo complete clinical examination ,dermascopic examination and diagnostic tests including hematological, biochemical,histopathologic tests and to undergo treatment ❑

Signature/thumb impression
Patient's Name and Address:

Signature of the investigator
Study Investigator's name:
Dr.Priyadharsini J

Gaurdian's name:
Relation to the patient :

ஆய்வு தகவல் தாள்

ஆராய்ச்சியின் தலைப்பு : தழும்பு வழுக்கை பற்றிய மருத்துவ, டெர்மாஸ்கோபி மற்றும் மெய்ம்மி நோயியில் ஆய்வு.

ஆய்வாளர் : மரு. பிரியதர்ஷிணி.ஜெ

பங்கேற்பாளர் : வயது : பாலினம் :

ஆராய்ச்சி மையம் : தோல்நோய் துறை,
இராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை.

இந்த ஆய்வில் பங்கேற்பதற்காக தாங்கள் அழைக்கப்படுகிறீர்கள். இந்த ஆவணத்தில் உள்ள தகவல்கள் தாங்கள் இந்த ஆய்வில் பங்கேற்க முடிவு செய்துக் கொள்ள உதவும். இதில் ஏதேனும் சந்தேகம் இருந்தால் வெளிப்படையாக கேள்விகளைக் கேட்டு தெரிந்துக் கொள்ளலாம்.

நாங்கள் இராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் தழும்பு வழுக்கை பற்றிய மருத்துவ, டெர்மாஸ்கோபி மற்றும் மெய்ம்மி நோயியல் ஆய்வை நடத்துகிறோம்.

அதற்கு உங்கள் பங்களிப்பு எங்களுக்கு பெரிதும் உதவக்கூடும்.

இந்த ஆய்வின் நோக்கம்:

இவ்வாராய்ச்சியில் தங்களிடையே அடிப்படை மற்றும் உங்களுடைய நோய் குறித்த விரிவான கேள்விகள் கேட்கப்படும். பின்னர் நீங்கள் மருத்துவப் பரிசோதனைக்கு உட்படுத்தப்படுவீர்கள். பின்பு தோல் சம்பந்தமான வெளிப்பாடுகள் குறித்து மருத்துவப் புகைப்படம் எடுக்கப்படும்.

அனைவரிடமும் இரத்தம் மாதிரி பெறப்பட்டு அது வழக்கமான இரத்தப் பரிசோதனைகளும் (VCTC, VDRL, CBC, LFT, RFT மற்றும் தேவைப்படுகின்ற நோயாளிகளுக்கு ANA, PUS C/S, Mantoux, Scraping பரிசோதனையும் செய்யப்படும். அனைவருக்கும் தழும்பு வழுக்கை உள்ள இடத்தில் டெர்மாஸ்கோபி மற்றும் அவ்விடத்திலிருந்து தோல் எடுக்கப்பட்டு உயிர்திடசுப் பரிசோதனைக்கு அனுப்பப்படும்.

தங்களது மருத்துவ சிகிச்சை குறித்த தகவல்கள் இரகசியமாக பாதுகாக்கப்படும். ஆய்வின் போதோ அல்லது முடிவுகளை வெளியிடும் போதோ தங்களது பெயரையோ, அடையாளங்களையோ வெளியிடமாட்டோம் என்பதை தெரிவித்துக் கொள்கிறோம்.

இந்த ஆய்வில் பங்கேற்பது உங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆய்விலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம். இந்த ஆய்வில் பங்கேற்காவிட்டாலும் நீங்கள் வழக்கமான சிகிச்சையை தொடர்ந்து பெறலாம்.

இந்த ஆய்வின் முடிவு தங்களுக்கு ஆய்வின் இறுதியிலோ அல்லது ஆய்வின் போதிலோ தெரியப்படுத்தப்படும்.

ஆய்வாளர் கையொப்பம்

பங்கேற்பாளர் / பாதுகாவலர் கையொப்பம்

தேதி :

சுய ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு : தழும்பு வழுக்கை பற்றிய மருத்துவ, டெர்மாஸ்கோபி மற்றும் மெய்ம்மி நோயியில் ஆய்வு.

பெயர் : வயது : தேதி : உள்நோயாளி எண் :

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக அறிந்து கொண்டேன். எனது சந்தேகங்கள் அனைத்திற்கும் தகுந்த விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுயநினைவுடன் பங்கு கொள்ள சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது.

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்து கொண்டேன்.

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில்தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆய்வில் கலந்து கொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர் இன்ஸ்டிட்யூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால் பகிர்ந்து கொள்ளலாம் என சம்மதிக்கிறேன்.

இந்த ஆய்வின் முடிவுகளை வெளியிடும்போது எனது பெயரோ, அடையாளமோ வெளியிடப்பட்டாது என அறிந்து கொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்று கொண்டேன். இந்த ஆய்விற்காக இரத்தப் பரிசோதனைகளும் (VCTC, VDRL, CBC, LFT, RFT, Dermascopy, உயிர் திடசுப்பரிசோதனை, மேலும் தேவைப்பட்டால் ANA, PUS C/S, Mantoux, Scraping பரிசோதனையும் செய்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை தொடர்பு கொள்ள வேண்டும் என அறிந்து கொண்டேன்.

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்றும் தெரிவிக்கிறேன் என்று புரிந்து கொண்டேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்றும் தெரிந்து கொண்டேன்.

பங்கேற்பாளர் / பாதுகாவலர் கையொப்பம் தேதி :

ஆய்வாளர் கையொப்பம் தேதி :

URKUND

Urkund Analysis Result

Analysed Document: Cicatricial alopecia p- Copy.docx (D31079314)
Submitted: 10/6/2017 3:09:00 PM
Submitted By: heidi.doc90@gmail.com
Significance: 1 %

Sources included in the report:

http://actasdermo.org/en/scarring-alopecia/articulo/S1578219012001709/

Instances where selected sources appear:

1

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301A
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CERTIFICATE OF APPROVAL

To
Dr.Priyadharsini.J.
II Year Post Graduate in MD DVL
Department of Dermatology
Madras Medical College
Chennai 600 003

Dear Dr.Priyadharsini.J.,

The Institutional Ethics Committee has considered your request and approved your study titled **"CLINICAL, DERMASCOPY AND HISTOPATHOLOGICAL STUDY OF CICATRICIAL ALOPECIA " NO. 08122016.**

The following members of Ethics Committee were present in the meeting hold on **14.12.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.A.Rajendran,MS, Prof. of Surgery,MMC,Ch-3	: Member
6.Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch	: Member
7.Prof.Baby Vasumathi,MD.,Director, Inst. of O & G	: Member
8.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
9.Prof.R.Padmavathy, MD, Director,Inst.of Pathology,MMC,Ch-3	: Member
10.Prof.S.Mayilvahanan,MD,Director, Inst. of Int.Med,MMC, Ch-3	: Member
11.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

21.12

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

CERTIFICATE –II

This is to certify that this dissertation work titled **"CICATRICIAL ALOPECIA - CLINICAL, DERMASCOPIC AND HISTOPATHOLOGICAL STUDY"** of the candidate **Dr.PRIYADHARSINI .J** with registration Number **201530006** for the award of **M.D DERMATOLOGY, VENEREOLOGY & LEPROSY** in the branch of **XX**. I personally verified the urkund .com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **1 percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal